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A CLINICAL EVALUATION OF DECORTICATION

DAVID H. WATERMAN, M.D., SHELDON E. DOMM, M.D., AND WILLIAM K. ROGERS, M.D. (BY INVITATION)

KNOXVILLE, TENN.

THE freeing of an imprisoned lung to obliterate persistent pleural space, and, more important, to restore pulmonary function, has long been a surgical challenge. The first attempts by Fowler[1] and by Delorme[2, 3] in the 1890's were admittedly unsatisfactory as to surgical technique and results. Later, others,[4] particularly Eggers,[5] applied surgical principles to the operation that are still pertinent and valid. Without antibiotics and the present surgical safeguards, especially in anesthesia and blood replacement, the operation remained a most formidable one. Under Churchill's[6] direction in the Mediterranean Theater during the last war, Burford, Parker, and Samson[7] and others re-pioneered the field and introduced techniques and produced results that have revived interest in the procedure and made it accepted by all thoracic surgeons.

Quite naturally the first problem attacked by Fowler and by Delorme was empyema. Pleural infection continued to be the predominant challenge through the First World War and, in fact, until the Second. The dangers of a persistent pleural infection were well recognized. Obliteration of the space by various types of thoracoplasty, while accomplishing this end, left much to be desired. Decortication was applied in an attempt to obtain a more satisfactory surgical answer, primarily with the object in mind of obliterating pleural space.

The other pressing problem was hemothorax, particularly brought to the fore because of the large number of chest wounds of the past war. The concept of restoring a lung to function actually was initiated during this period when it was demonstrated that decortication would permit the injured soldier

From the Departments of Surgery of the Fort Sanders Presbyterian Hospital and the East Tennessee Tuberculosis Hospital, Knoxville, Tenn.

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to return to duty in a high percentage of instances. The technique was immediately applied widely in civilian practice as well, with comparable good results. Infection became less and less of a problem, though not infrequently complicating the hemothorax.

This was the era of pneumothorax therapy. Pneumothorax was theoretically and, in most cases practically, a reversible procedure. In too many, however, it was not, and space either persisted or "false re-expansion" resulted. In the first instance, decortication was seized upon as the solution to the problem, first by Mulvihill and Klopstock,[8] and then by many others. The results were most gratifying, with satisfactory obliteration of the space. Selective decortication,[9] leaving a plaque of peel over the area of disease, was used to render the danger of reactivation of foci less likely. Residual tuberculous disease in the lung at this time was rarely resected.

MATERIAL

In the past eleven years, the authors have had an experience with 172 cases in which pulmonary decortication was performed either as a primary or a secondary procedure. Eighty-five per cent of these have been performed in the past six years. Of the 172 cases, 140 were operated upon by the authors, and 32 by other staff members of the East Tennessee Tuberculosis Hospital. It is the purpose of this report to analyze this group of cases as to the type of operative procedure performed, the indications for such procedures, the complications encountered, the mortality occurring, and the results obtained. Clinical evaluation in all cases, and pulmonary function studies, including bronchospirometry in a selected group, have been used to assay the results.

OPERATIVE PROCEDURES

The types of operative procedures performed in the 172 cases are listed in Table I. Simple decortication alone was performed in 70 cases. Lobectomy was combined with decortication in 49, and segmental resection in 38. Included in this last figure are both dissection segmental resections and wedge procedures, giving a total of 87 patients in which pulmonary tissue was extirpated in conjunction with the decortication. Thoracoplasty was done in 10 patients having decortication, 8 of these being performed in connection with a relaxing thoracoplasty following resection. In such cases the pleural space was entered, the underlying lung decorticated, and the thorax tailored to fit the remaining space. The other 2 cases were in conjunction with Schede thoracoplasty done for long-standing empyema.

TABLE I. OPERATIVE PROCEDURES
172 CASES

Decortication alone	70
with lobectomy	49
with segmental resection	38
with thoracoplasty	10
with diaphragmatic herniorrhaphy	3
with plication of diaphragm	2

A diaphragmatic hernia was repaired in 3 cases, the decortication being a primary procedure and the hernia an incidental finding in one patient, and the reverse in the other 2.

In 2 cases of a high diaphragm, resulting from previous phrenic interruption remaining permanent rather than temporary, plication was performed to restore the diaphragm to a normal level and to permit the lung to expand in an adequate space. It was of interest that in both of these patients a markedly attenuated diaphragm, apparently showing no function whatsoever on fluoroscopy preoperatively, was restored to a limited excursion following the plication procedure.

INDICATIONS

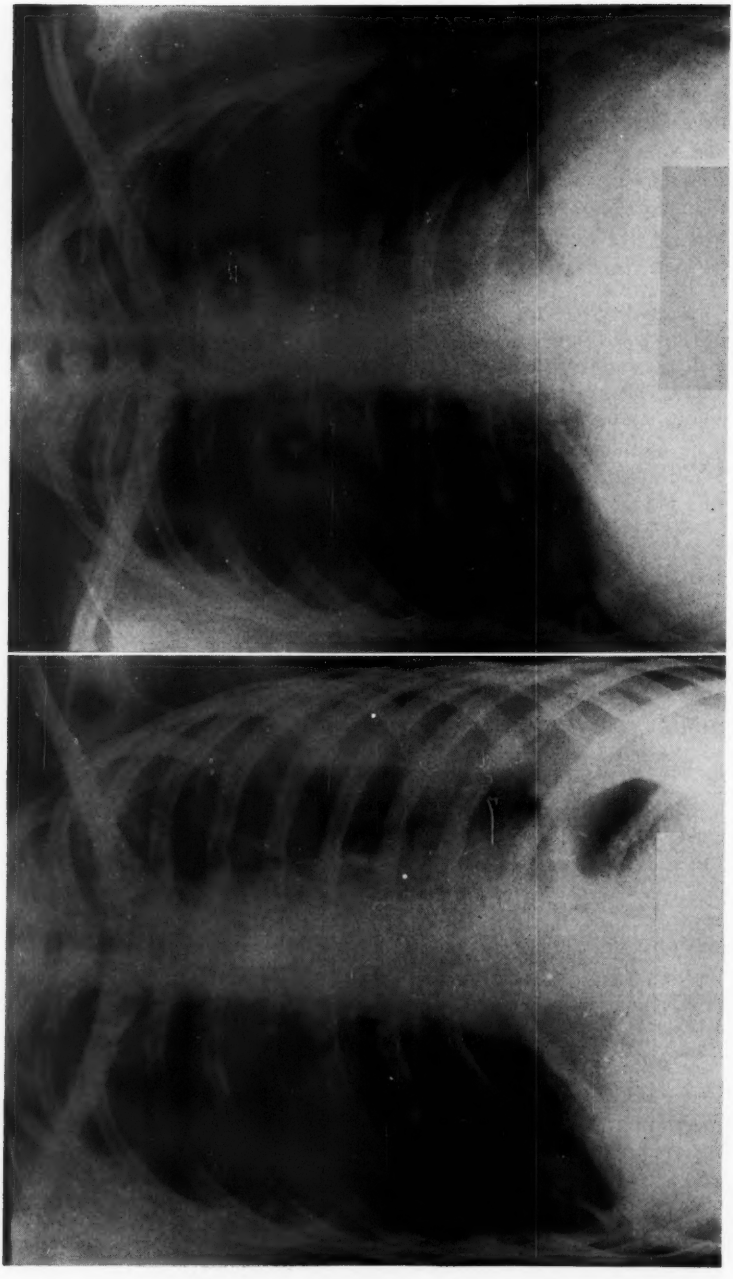
It has been our feeling that the indications for decortication are far wider than the three already enumerated of empyema, hemothorax, and unexpandable pneumothorax lung with persistent space. Essentially, of course, the basic indication is an imprisoned lung which can be expanded either to restore function and/or to obliterate pleural space if such remains. We have grouped the disease conditions indicating decortication in three categories—tuberculous, nontuberculous, and malignant.

TABLE II. INDICATIONS—TUBERCULOUS
115 CASES

Unexpandable pneumothorax lung		39
Unilateral—with space	30	
Unilateral—with hemorrhage into space	2	
Bilateral—with space	2	
Unilateral—without space ("False re-expansion")	5	
Restricted lobe(s) limiting expansion at resection		55
with expanded pneumothorax	12	
with "false re-expansion"	6	
without previous pneumothorax	37	
Restricted lobe at thoracoplasty for postresection residual space		8
Tuberculous effusion		6
Tuberculous empyema		7
with bronchopleural fistula	5	
without bronchopleural fistula	2	

Tuberculous Disease.—Table II lists the indications for decortication in tuberculous disease in our 115 cases. In unexpandable pneumothorax lung, the results have been most gratifying. Thirty-nine patients in our series had previous pneumothorax, 30 having the classical indication of unilateral unexpandable lung with persistent pleural space. Two of these had hemorrhage into the space which remained sterile, and essentially had their procedures done as emergencies because of exsanguination.

Two patients had unexpandable lung on both sides. One of these was so short of breath that he was unable to go to the bathroom without assistance.



A. *B.*

Fig. 1.—Case W. R., a 28-year-old white man. *A*, Preoperative x-ray. *B*, Postoperative x-ray following bilateral decortication.

After decortication of one side he became ambulatory and, after the second decortication, he returned to full activity of strenuous character. This patient was a 28-year-old white man who had pneumothorax on the left side from March, 1949 until decortication on Nov. 14, 1951, and pneumothorax on the right from March, 1950, until decortication and resection of the posterior segment of the right upper lobe and a wedge resection of a small portion of the anterior segment on March 18, 1953. His pre- and postoperative x-rays are shown in Fig. 1.

So-called "false re-expansion" was a not uncommon occurrence in pneumothorax cases. The presence of a peel prevented the re-expansion of the lung, and the space was obliterated by shift of the mediastinum, elevation and fixation of the diaphragm, and contraction of the chest wall. Function was seriously impaired. In each case in this category subjected to operation, the condition has proved reversible to an appreciable extent in spite of its persistence for a number of years. Bronchospirometric data, given later, have corroborated this impression. The average duration of "false re-expansion" in our series has been four and one-half years, the longest being seven years.

The largest single group in our series is that of a restricted lobe or lobes limiting expansion at resection. Fifty-five cases have fallen into this category, 12 of the patients having had previous expanded pneumothorax with residual peel restricting the lung, 6 "false re-expansion," and 37 no history of pneumothorax. In the latter, the peel was encountered at surgery and was often unsuspected. In many of the resection cases, decortication was of course a secondary procedure. It is a cardinal principle that obliteration of residual pleural space following resection should take place as quickly as possible. Obviously a restricted lobe cannot do this.

When pleural space has persisted after resection and thoracoplasty has been indicated, we have found that decortication of the underlying lung frequently reduces the amount of thoracoplasty necessary.

Tuberculous effusion may result in marked constriction and infolding of the lung on the affected side, with fixation and loss of function of the diaphragm. Decortication can correct this situation. Six of our cases fall into this group. Tuberculous empyema, either with or without bronchopleural fistula, is admittedly more rare than heretofore. We have had seven instances, 5 with fistula and 2 without, in which decortication has been done. Such decortications are more difficult, as there may be no satisfactory line of cleavage between the visceral pleura and the peel. The closure of the bronchopleural fistula frequently results if the space can be obliterated promptly.

Nontuberculous Disease.—The conditions for which decortication was performed in the nontuberculous group are given in Table III.

Hemothorax cannot always be managed by aspiration and by enzymatic débridement. Of the 21 cases in our series, 17 were traumatic, 3 spontaneous, and one postoperative. Three of the traumatic patients had a thick calcified

plaque restricting the lung following gunshot wounds fourteen, nineteen, and twenty-two years before. In each instance the lung was found to be readily expansile following the removal of the plaque.

TABLE III. INDICATIONS—NONTUBERCULOUS
47 CASES

Hemothorax		21
traumatic	14	
spontaneous	3	
calcified	3	
postoperative	1	
Chronic empyema		4
Chronic empyema with calcification		2
Empyema following resection		4
Empyema following ruptured lung abscess		2
Spontaneous pneumothorax		7
Bronchiectasis		3
Bullous emphysema		1
Partial agenesis of lung		1
Diaphragmatic hernia		1
Chylothorax		1

It is of particular interest that the patient in whom the calcified plaque was present for twenty-two years also had an unsuspected tumor encountered at operation. The presence of the bronchogenic carcinoma was completely masked by the heavy plate of calcium.

Chronic empyema is becoming considerably less common with the lowered incidence of acute empyema and the better handling of cases by thoracic surgeons. Four in our series fell into this category. Two had long-standing empyema with a calcified plaque that had to be removed to permit expansion of the underlying lung. In 4 patients, the empyema followed resection and, in two instances, ruptured lung abscess.

A peel can form rather rapidly if spontaneous pneumothorax persists, particularly in the presence of fluid. Seven patients in our series required decortication when other methods of expanding the pneumothorax were ineffective. In three instances of resection for bronchiectasis, a peel was encountered over nonresected areas that prevented adequate re-expansion. The same was true in one case of bullous emphysema. In the case of partial agenesis of the lung, a thick fibrous peel covered the entire remaining lung tissue. The middle lobe consisted only of a bronchiectatic nubbin which was resected. A peel was also encountered in 3 patients with diaphragmatic hernia, evidently due to inflammatory reaction around the sac. It was necessary to free the lung to restore anatomic relationship. In the case of chylothorax, a lymphoma had eroded the thoracic duct. At exploration a thick peel was encountered.

Malignant Disease.—Table IV lists the 10 cases of malignancy for which decortication was performed. The first of these was done in 1945 for uncontrollable pleural effusion due to metastatic malignancy. Obliteration of the space and control of the fluid resulted. In the other 4 similar cases, the same

TABLE IV. INDICATIONS—MALIGNANT
10 CASES

Uncontrollable pleural effusion secondary to metastatic malignancy	5
Restricted lobe(s) secondary to bronchogenic carcinoma	5

result was obtained. We would not imply that decortication should be the procedure of choice in all cases of malignant effusion. Routinely we do pleural biopsy now on all cases of effusion in which the diagnosis has not been established. However, when intrapleural nitrogen mustard and intrapleural isotopes have been ineffective, we have been able by decortication to obliterate the pleural space and obviate the necessity for frequent repeated thoracenteses.

In 5 patients, a peel has been encountered on exploration for bronchogenic carcinoma. In 2 cases, lobectomy was done electively because of low pulmonary reserve, the remaining lobe or lobes being decorticated to fill the space. In the other three instances, a peel was encountered and decortication done of necessity to permit further exploration before inoperability was determined. Needless to say, such a condition is not an indication for decortication in itself.

SURGICAL TECHNIQUE

Although it is beyond the scope of this paper to describe the operative technique in detail, it is pertinent to enumerate certain surgical maneuvers which we feel are important in preserving and restoring pulmonary function, and which would aid in the interpretation of the complications, the mortality, and the results given in this paper.

In our hands an intercostal incision has been the most satisfactory and has been used in practically all cases. We believe that more satisfactory closure can be obtained, that less postoperative pain occurs, and that better chest wall function results.

Removal of the parietal pleura as well as the visceral peel is always done if sufficient parietal pachypleuritis is present to interfere with return of chest wall motion.

The diaphragm is mobilized to its origin and decorticated, if possible, to permit maximum return of excursion.

All adhesions are freed, fissures developed, and infolding of the lung corrected by removal of overlying webs of peel.

The phrenic nerve must obviously not be injured in the decortication of the mediastinum.

To prevent prolapse, the lung is tacked into optimal position with several catgut sutures, usually at the apex and often at the fissures, before closing the chest. Especially important where raw surfaces are present is the tacking down of the edge of the lower lobe to the diaphragm in the costophrenic sulcus to interpose lung tissue between the surfaces of the diaphragm and the chest wall so that the sulcus will not become obliterated and the pistonlike action of the diaphragm impaired.

Air leaks should be avoided or closed insofar as possible so that maximum prompt re-expansion of the lung can be achieved. Adequate active suction, with replacement of drainage tubes if necesary, is of extreme importance.

Selective breathing exercises, begun prior to operation and continued vigorously postoperatively, even for several months, can do much to restore normal chest wall expansion on the operated side.

It is obvious that a good final functional result must depend greatly upon the technical success of the surgical procedure.

COMPLICATIONS

Twenty-five complications were encountered in the 172 operated cases. Twelve of these occurred in patients with concomitant pulmonary resection, as shown in Table V. Of these 87 cases, 6 had persistent pleural space requiring the insertion of additional tubes before such space was obliterated. Empyema with bronchopleural fistula resulted in 5 cases. It should be pointed out that these complications were essentially those of the resection and not of the decortication. The same can be said for the tuberculous spread that occurred in the other additional case. This was in a patient undergoing resection under thoracoplasty.

TABLE V. COMPLICATIONS
87 CASES

DECORTICATION WITH RESECTION	
Persistent pleural space	6
Empyema and bronchopleural fistula	5
Tuberculous spread	1
Total	12 (13.8%)

The other thirteen complications were found in the 85 cases in which no resection was combined with the decortication. These are listed in Table VI. There were two persistent air leaks necessitating reinsertion of tubes. Two patients had empyema without demonstrable fistula, both being handled without surgical intervention.

TABLE VI. COMPLICATIONS
85 CASES

DECORTICATION WITHOUT RESECTION	
Persistent air leak	2
Empyema without apparent fistula	2
Postoperative hemorrhage	2
Subcutaneous emphysema, severe	2
Phrenic nerve injury	2
Persistent pleural space	1
Chest wall tuberculosis	1
Tear of superior vena cava	1
Total	13 (15.3%)

Serious postoperative hemorrhage occurred in 2 patients. One, not in our own series, had considerable disease in the underlying lung so that space obliteration was not achieved. Hemorrhage into the space was uncontrolled and death resulted. In the other instance, a thick calcified peel present fourteen years had been removed with some difficulty. Re-exploration of the patient to control the bleeding was necessary. Two patients had severe subcutaneous emphysema requiring superficial skin incisions.

Another complication was that of an inadvertent permanent injury to the phrenic nerve in decorticating the mediastinum. This also occurred in a second patient, but end-to-end anastomosis of the nerve was successful and diaphragmatic function resulted.

One pleural space persisted at the apex; a small thoracoplasty was necessary. Chest wall tuberculosis resulted after decortication in a previously unsuspected tuberculous empyema with routine negative cultures. One of the other fatalities of the series resulted from a tear of the superior vena cava in removing an extremely thick peel.

The cases of hemorrhage and the permanent phrenic paralysis were the only serious complications of this group.

The incidence of complications was 13.8 per cent in the cases of resection with decortication, and 15.3 per cent in the rest of the series.

MORTALITY

There were four deaths, giving a mortality of 2.3 per cent, in the 172 cases listed in Table VII. The case of operative hemorrhage was the tear of the superior vena cava mentioned under "Complications." The postoperative hemorrhage, also described under "Complications," was in the case of bilateral unexpandable pneumothorax in which decortication was unsuccessful because of severe underlying disease, and hemorrhage could not be satisfactorily controlled.

The other two deaths resulting from pulmonary insufficiency were salvage cases having extremely poor over-all pulmonary function tests prior to surgery. They represent poor operative selection of patients.

TABLE VII. MORTALITY
172 CASES

Operative hemorrhage	1
Postoperative hemorrhage	1
Pulmonary insufficiency	2
Total	4 (2.3%)

CLINICAL RESULTS

The results of the 139 cases in whom adequate follow-up was obtained are shown in Table VIII. The criteria by which the patients were classified were both subjective and objective. The great majority of the patients reported that they were able to breathe much better following the procedure than before. They also reported that their exercise tolerance was greater.

The patients were also evaluated as to whether they were incapacitated before and able to return to full activity after the decortication. At physical examination particular attention was paid to the quality of the breath sounds,

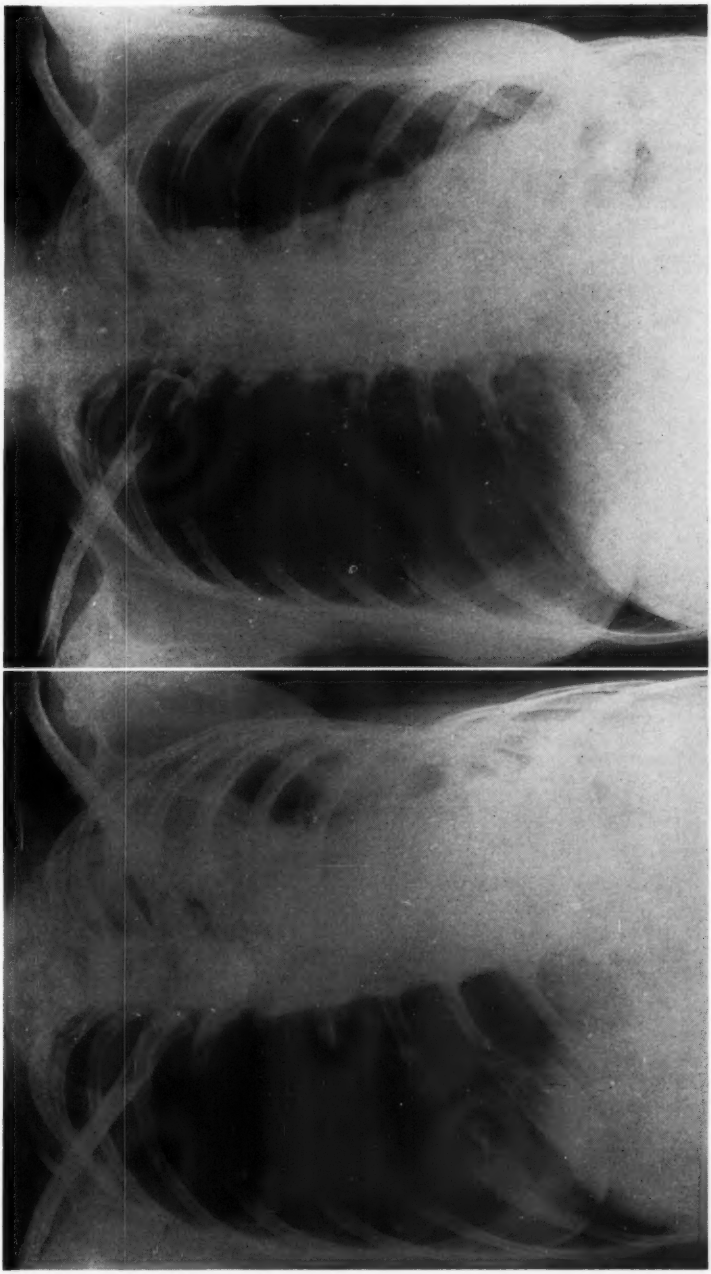
TABLE VIII. CLINICAL RESULTS
139 CASES (WITH ADEQUATE FOLLOW-UP)

	GOOD	FAIR	POOR
Decortication without resection	50	12	7
Decortication with resection	58	8	4
Total	108 (77.7%)	20 (14.4%)	11 (7.9%)

TABLE IX. BRONCHOSPIROMETRY

CASE	DIAGNOSIS	OPERATION	TIMING OF STUDIES	RESTING VENTILATION OPERATED SIDE % OF TOTAL	VITAL CAPACITY OPERATED SIDE % OF TOTAL	O_2 UPTAKE OPERATED SIDE % OF TOTAL	VENTILATORY EQUIVALENT $\frac{\text{VENTIL. IN LITERS}}{O_2 \text{ UPTAKE IN C.C.}} \times 100$ OPERATED SIDE	VENTILATORY EQUIVALENT WELL SIDE
1. R. P.	Pulm. tbc. MAC*	Pulm. decort., left	Preop.	15	4	9	2.5	1.3
	Pneumothorax, lt.		Postop.	40	25	40	1.3	1.3
	"False re-expansion"		9 mo.					
2. H. C.	Pulm. tbc. MAC	Pulm. decort., right;	Preop.	26	17	19	6.3	4.4
	Pneumothorax, rt.	wedge, RUL	Postop.	40	34	52	2.0	3.2
	"False re-expansion"		25 mo.					
3. E. V.	Pulm. tbc. FAC†	Pulm. decort., left	Preop.	35	27	23	3.0	1.7
	Pneumothorax, lt.	(previous plom-	Postop.	44	44	41	1.1	1.0
	"False re-expansion"	bage right)	(left)					
			8 mo.					
4. A. H.	Pulm. tbc. MAC	Pulm. decort., right;	Preop.	47	31	43	1.2	1.0
	Pneumothorax, rt.	seg. RUL	Postop.	51	40	54	2.1	2.3
	"False re-expansion"		8 mo.					
5. L. M.	Pulm. tbc. FAC	Pulm. decort., left;	Preop.	32	16	? (air leak)	?	?
	Pneumothorax, lt.	seg. LUL	Postop.	36	33	43	1.3	1.7
	"False re-expansion"		19 mo.					
6. D. B.	Pulm. tbc. MAC	Pulm. decort., left;	Preop.	25	18	20	2.5	1.9
	Pneumothorax space	seg. LUL	Postop.	26	27	33	1.0	1.4
			36 mo.					
7. C. B.	Pulm. tbc. MAC	Pulm. decort., left;	Preop.	41	35	30	2.6	1.6
	Pneumothorax, lt.	wedge LUL	Postop.	49	41	36	2.4	1.4
	Re-expanded							
Mean			Preop.	31.6	21.1	24.0	3.0	2.0
			Postop.	40.9	42.0	42.5	1.6	1.7

*MAC—Moderately advanced, chronic.
†FAC—Far advanced, chronic.



A. *B.*

Fig. 2.—Case R. P., a 39-year-old white woman. *A*, Preoperative x-ray. *B*, Postoperative x-ray following left decortication.

and their intensification on deep breathing. The extent to which motion of the chest wall returned was noted, as well as the widening of contracted interspaces and the correction of scoliosis. X-ray and fluoroscopy revealed whether the costophrenic angles were satisfactory and whether the excursion of the diaphragm was adequate.

On the basis of these studies, the results were classed as good, fair, or poor. Fifty of the cases in which decortication alone or decortication plus some procedure other than resection was done were classed as a good result, 12 as a fair result, and 7 as a poor result. Four of these poor results were the cases reported under "Mortality." Another was a decortication for metastatic malignancy in which a small empyema persisted.

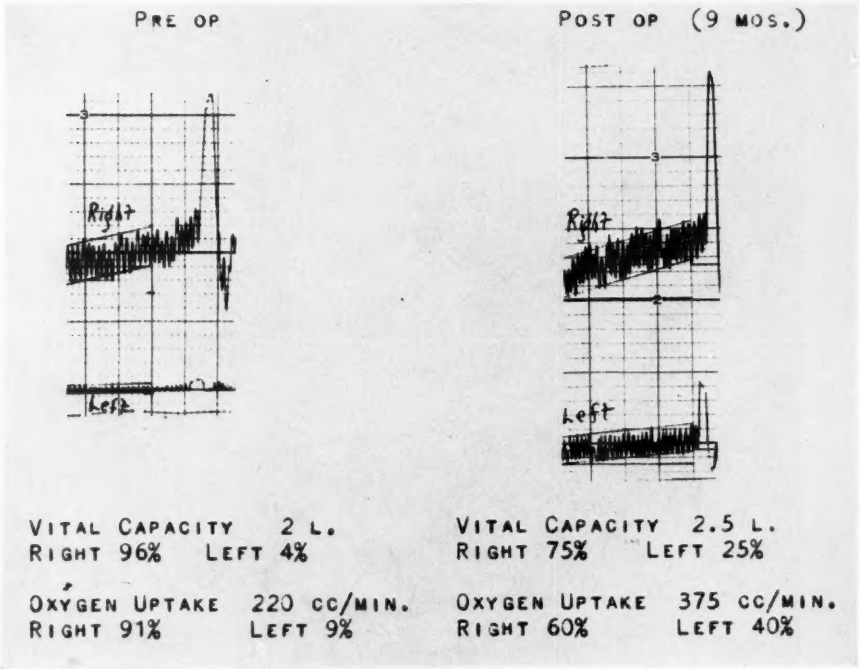


Fig. 3.—Case R. P., a 39-year-old white woman. Preoperative and postoperative bronchospirometric tracings and data.

Fifty-eight of the patients having a decortication plus a resection were classed as good, 8 as fair, and 4 as poor.

Of the 139 cases followed up, 108 or 77.7 per cent had a good result, 20 or 14.4 per cent a fair result, and 11 or 7.9 per cent a bad result.

Pulmonary Function Study Data.—Although the subject of this paper is the clinical evaluation of pulmonary decortication, the corroborative value of pulmonary function studies is assumed on the basis of the work of Wright,[10] Patton, Watson and Gaensler[11] and others. When feasible, we have done overall ventilatory pulmonary function and bronchospirometric studies pre- and

A. *B.*

Fig. 4.—Case H. C., a 30-year-old white man. *A*, Preoperative x-ray. *B*, Postoperative x-ray following right decortication.

postoperatively. Seven such cases, all tuberculous, having technically satisfactory tracings are presented. Five of these fall into the so-called "false re-expansion" group in which we are particularly interested.

The results show significant improvement by the operated side in every category, that is, resting ventilation, vital capacity, oxygen uptake, and ventilatory equivalent.

Two cases are presented for illustration.

CASE REPORTS

CASE R. P.—A 39-year-old white woman. Pulmonary tuberculosis, moderately advanced (small cavity LUL). Pneumothorax, left, three years, with "false re-expansion" for four years. Phrenemphraxis, left, regenerated.

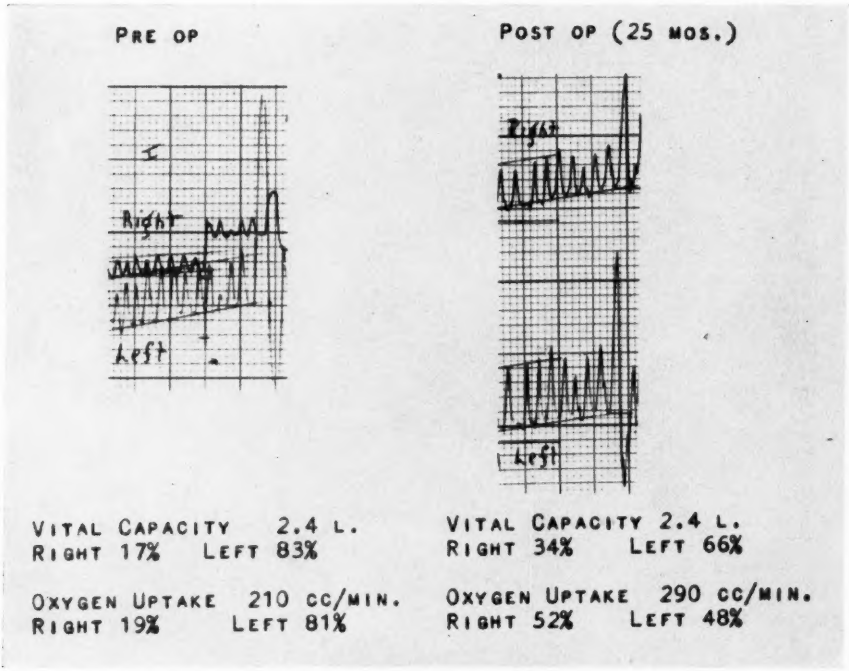


Fig. 5.—Case H. C., a 30-year-old white man. Preoperative and postoperative bronchospirometric tracings and data.

Operation: Pulmonary decortication, left. Fig. 2 shows the pre- and postoperative x-rays, and Fig. 3 the pre- and postoperative bronchospirometric tracings and data.

Comment: There was significant increase in ventilation and oxygen uptake on both sides, relatively greater on operated side, following decortication. It is of interest that vital capacity and maximum breathing capacity are both 91 per cent of estimated normal, one year postoperatively.

CASE H. C.—A 30-year-old white man. Pulmonary tuberculosis, moderately advanced (small cavity RUL). Pneumothorax, right, three and one-half years with effusion and "false re-expansion" for two and one-half years.

Operation: Pulmonary decortication, right, plus wedge resection nodule right upper lobe, posterior segment. The pre- and postoperative x-rays are shown in Fig. 4, and the bronchospirometric tracings and data in Fig. 5.

Comment: There was relative increase in ventilation on operated side, and over-all increase in oxygen uptake with relatively greater increase on operated side, following decortication.

SUMMARY

1. A series of 172 cases of pulmonary decortication is presented. Seventy patients had decortication alone, 87 decortication with resection, 10 with thoracoplasty, 3 with diaphragmatic herniorrhaphy, and 2 with plication of the diaphragm.

2. The disease conditions indicating decortication in tuberculosis included unexpandable pneumothorax lung, either with or without space. Eleven cases of "false re-expansion" are of particular interest. Rehabilitation by decortication of a case of bilateral unexpandable lung is presented. The largest group operated upon was composed of those with a restricted lobe or lobes limiting expansion at resection. A limited number of restricted lobes found at thoracoplasty were also decorticated. Tuberculous effusion and tuberculous empyema were additional indications.

3. In nontuberculous disease, hemothorax, either traumatic, spontaneous, or postoperative, was a frequent indication. The presence of calcium in the organized hemothorax is not a contraindication. Three such cases are reported of fourteen, nineteen, and twenty-two years' duration. Chronic empyema, with or without calcification, and empyema following resection or lung abscess, indicated decortication in a number of cases in the series. Additional cases of spontaneous pneumothorax, bronchiectasis, bullous emphysema, partial agenesis of the lung, diaphragmatic hernia, and chylothorax are enumerated.

4. Malignant indications were found where pleural effusion was uncontrollable or peel prevented the expansion of the remaining lobes following elective lobectomy in bronchogenic carcinoma.

5. The incidence of complications was 13.8 per cent in the cases in which decortication was combined with resection, and 15.3 per cent in cases without resection.

6 There were four deaths in the series, one from operative hemorrhage, one from postoperative hemorrhage, and two from pulmonary insufficiency.

7. One hundred and thirty-nine cases with adequate follow-up were evaluated as to results. Seventy-seven and seven-tenths per cent had good results, 14.4 per cent fair results, and 7.9 per cent poor results. Deaths were included in the poor results.

8. Bronchospirometric studies giving technically satisfactory tracings on a group of 7 tuberculous patients, 5 falling into the group of "false re-expansion," are presented. Significant improvement was shown by the operated side in resting ventilation, vital capacity, oxygen uptake, and ventilatory equivalent.

CONCLUSIONS

1. Decortication can be applied widely in a number of conditions, tuberculous, nontuberculous, or malignant, stemming from imprisoned lung either with or without a pleural space. Mortality and complication rates are acceptable.

2. "False re-expansion" of a lung following pneumothorax is a valid indication for decortication, as in intractable malignant effusion.

3. The final results of decortication depend not upon the disease for which decortication was done, but upon the two factors of the functioning potential of the underlying imprisoned lung, and the technical success of the surgical procedure.

The cooperation given by Dr. Perry M. Huggin, Medical Director, and Dr. Charles L. Butler, Pathologist, of the East Tennessee Tuberculosis Hospital is gratefully acknowledged.

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DISCUSSION

DR. ALLAN STRANAHAN, Albany, N. Y.—We too have been interested in pulmonary function studies before and after decortication. I would like to present one case which illustrates the value of preoperative bronchograms in deciding whether or not one should do a resection with a decortication.

(slide) This 60-year-old white man's present illness began in 1910 when he had pneumonia followed by an empyema on the left. At that time the left pleural cavity was drained and bismuth paste was instilled in the pleural space at the time of drainage. He gradually regained his stamina and was apparently well until 1950 when he again had symptoms of sepsis, chills and fever, night sweats, increased shortness of breath, with cough productive of large amounts of thick yellow sputum. The left pleural cavity was tapped and *Staphylococcus aureus* was obtained in culture.

(slide) A bronchogram was made to identify the extent of pulmonary disease and this showed fairly complete filling of the smaller branch bronchi of the upper lobe with rather marked distortion and contraction of the lower lobe segments. These findings indicate pleural parenchymal fibrosis with scarring of the lung, and under ordinary circumstances are an indication for pulmonary resection in conjunction with decortication.

(slide) The poor pulmonary function studies and the patient's history of dyspnea indicated that he was a severe cardiopulmonary cripple. The maximum breathing capacity was only 37 L. per minute. The lung volume studies showed a high residual air, indicating considerable trapping of air and probable irreversible pulmonary emphysema. The bronchospirometry showed a poorly functioning left lung with some discrepancy of oxygen consumption and diminished ventilation. Accordingly, it was felt that the operation of choice in this particular patient should be that of decortication alone, rather than decortication and pulmonary resection, recognizing the fact that we would be leaving residual pulmonary disease.

(slide) Decortication was done, and this slide shows the bismuth paste which had been in the pleural cavity for forty years and also the cortical peel which was removed.

(slide) The postoperative result was not dramatic. However, the infection was removed and the upper lobe following decortication expanded quite well. There were multiple small bronchial fistulas arising in the superior segment of the lower lobe which were simply closed with fine catgut. The patient was discharged from the hospital without complications and at the present time is doing well.

(slide) The postoperative pulmonary function studies showed a slight increase in the maximum breathing capacity. The residual volume decreased a slight amount. There was some improvement in the oxygen comsumption and ventilation of the left lung. We did not, however, feel that this helped this patient a great deal from the standpoint of pulmonary function.

There is one other point I would like to make which Dr. Waterman only briefly mentioned, that is the ease of decorticating a lung in a patient who develops an empyema following pneumothorax, either therapeutic or spontaneous. In our experience, these lungs are rather easy to decorticate in contradistinction to those in patients whose empyema develops primarily without preceding pneumothorax. This latter type of patient usually has a severe degree of pleuroparenchymal scarring which makes decortication difficult if not impossible. However, if the onset is that of a primary tuberculosis or nontuberculosis empyema then, with the pleuroparenchymal fibrosis that is present, decortication is very difficult.

DR. JOHN S. CHAMBERS, San Diego, Calif.—I enjoyed Dr. Waterman's paper very much and wish to direct the following remarks to his comments about decortication for neoplastic pleural effusion. In patients with pleural effusion, secondary to neoplasm both primary in the lung and metastatic, intercostal intubation and instillation of a suspension of talcum powder has been used with the idea of producing an obliterative pleuritis and obviating frequent chest taps. In 20 of 23 patients no further thoracenteses were necessary.

I believe these results are superior to those obtained with radioactive gold and that this procedure would make decortication for palliation unnecessary in neoplastic effusion.

DR. GABRIEL SELEY, New York, N. Y.—This is a timely subject and I want to thank Dr. Waterman for reintroducing it. I would like to stress the use of decortication in acute and subacute empyemas. We have seen an increasing number of acute empyemas due to resistant strains of microorganisms, especially hemolytic *Staphylococcus aureus.* We as chest surgeons are usually not consulted until after our medical colleagues have already used multiple aspirations and the conventional antibiotics have failed, due to loculations present in the lobar distribution of the empyema, or bacterial resistance to the drugs employed.

I am not talking about infected pleural fluid, but am trying to discuss a typical purulent pleuritis, a real empyema with pus. At this stage, enzymatic decortication is usually ineffective. Simple drainage may and often will result in chronic empyema, whereas a properly conducted decortication will prevent chronic empyema and restore lung function more rapidly and effectively. At the time of operation, one must pay particular attention to all the loculations, especially those beneath the lung and between the lobes. With the liberal use of blood transfusions and properly tested wide spectrum

antibiotics, complete decortication can be performed safely even in the extremely sick and toxic patient and, in fact, can be lifesaving. In addition, pulmonary foci, if present, can also be handled at the same time. Multiple tube suction should be instituted and continued for a long period, postoperatively.

A number of recent patients including adults and children treated in this manner have had complete and rapid restitution of normal anatomy and physiology.

DR. R. LEONARD KEMLER, Hartford, Conn.—First, I should like to commend Dr. Waterman for the excellent presentation of a very interesting subject. There was one line in one of the slides which interested me greatly; I believe it stated that he had used decortication for malignant pleural effusions in 5 cases. We have had experience in one case in which a patient came to us with a pleural effusion which contained malignant cells on several occasions; despite repeated tappings the effusion would recur every ten days. The patient was given x-ray therapy, radioactive gold and nitrogen mustard, and the pleural effusion continued to reaccumulate at a very rapid rate. The man was 55 years of age and in desperation. In order to make his remaining days as comfortable as possible, we attempted a decortication. To our surprise we were able quite easily to decorticate the lung, the pleural of which contained multiple tumor nodules. The parietal pleural surface was removed quite easily also. The patient lived for a year and one half afterward with no further tapping and with a very comfortable existence. I would like to hear Dr. Waterman's comments on the 5 cases in which he tried it.

DR. WILLIAM K. ROGERS, Knoxville, Tenn.—I would like to show one case of bilateral pneumothorax in which the lung was decorticated because of failure of re-expansion on both sides.

(slide) This is the x-ray at the time we saw the patient. He was unable to get out of bed to go to the bathroom by himself because he was so short of breath. The left side was attacked first.

(slide) This shows the peel that was removed on the left side.

(slide) This is the postoperative film after the left side had been decorticated.

(slide) This is the peel subsequently removed on the right.

(slide) This is the final film.

After the bilateral procedures had been carried out this individual was rehabilitated considerably. He now carries on one of the most strenuous occupations in eastern Tennessee, that of a Baptist preacher.

I would like to elaborate on one other point. As Dr. Waterman said, the purpose of decortication is twofold; to restore function and to obliterate pleural space. In going over one's cases, one may think he has had a poor result because the x-rays do not look good, or perhaps because the diaphragm does not move quite as well as he might wish. However, one must take into account what he wished to accomplish in the first place. It may not be necessary to have a perfect-looking x-ray; it may not be necessary to have a normally functioning diaphragm. If the patient is rehabilitated and if the pleural space has been closed and infection has been controlled, then decortication is certainly a very worth-while procedure regardless of the appearance of the x-ray.

DR. PAUL C. SAMSON, Lafayette, Calif.—I want to thank Dr. Waterman for reopening this subject. It became rather passé after the war and yet, as you can see, the indications have everwidened and I think it is worth while to reconsider them again. Likewise, I would like to thank him for his kind remarks toward your recorder.

I have two observations to make. The first is in relation to the use of some type of decortication in malignant disease. We have had a brief experience with this and I recall in particular 2 young children who had malignant disease and uncontrollable fluid to the extent that they were being aspirated every third or fourth day, as Dr. Kemler's patient was. Each had had x-ray therapy without success and, in each, a combination of decortication and parietal pleurectomy enabled the lung to re-expand; no further thoracenteses were necessary. Of course both youngsters died of their malignant disease but during the number of months that they lived they did not have to be aspirated.

I would take issue with Dr. Waterman on one point. He mentioned that there is no peel on the parietal pleura, if I heard correctly. I would remind you that there is a peel on the parietal pleura and, while the parietal pleura may thicken an infinitesimal amount, still and all we can decorticate the parietal peel and do so very frequently. I will agree, however, that in older and more chronic disease, techincally it is much easier to do a pleurectomy than it is to do decortication on the parietal side and therefore I agree insofar as he says pleurectomy is a preferable procedure. But there is a peel on the parietal pleural side, as one can tell if he examines these specimens carefully under the microscope.

Thank you very much. It was a good paper.

DR. JAMES E. DAILEY, Houston, Texas.—I merely wish to mention briefly that for quite a few years, in dealing with patients with nonresectable nonmalignant lesions and especially with those having associated pleural effusion, I have made it a habit to perform a parietal pleurectomy almost routinely with very gratifying results. The necessity for repeated aspiration stops with re-expansion and pleural symphysis and even more gratifying is the fact that these pleurectomized patients remain much more comfortable with respect to pain. In only very few cases have I taken the trouble to decorticate the visceral pleura. Parietal pleurectomy seems to suffice.

DR. A. R. VALLE, Detroit, Mich.—I would like to congratulate Dr. Waterman on this excellent presentation and his good results. However, I thoroughly agree with Dr. Samson that both the visceral and parietal peels should be removed. I feel that it is very important to remove the parietal peel because it is almost always the thicker and it immobilizes the rib cage and diaphragm, thus interfering with pulmonary function. Also, I think it is important to start active physical therapy soon after surgery since it aids in restoring the rib cage and lung to normal function. In our series of 278 nontuberculous patients, we obtained 91 per cent good results, 5 per cent fair results, and 4 per cent poor results. I must mention, however, that the majority of the patients in our series were young soldiers wounded in the Korean War, which no doubt influenced our results.

DR. RICHARD H. MEADE, Grand Rapids, Mich.—I was particularly interested in the use of this procedure in malignant disease. It seems quite logical that if you do a decortication and remove the parietal pleura, you will open up many channels of absorption. I should like to know what happens in cases where you do not do that. About eight months ago, I operated on a patient who had pleural effusion. We could not make a definite diagnosis of the source of it. At operation I found that she had an inoperable carcinoma of the lung. All I did was make a biopsy of the lung tumor. There has not been a drop of fluid since that time. I do not believe we know just what happens in such cases. Those of you who can remember many years ago when tuberculous peritonitis was seen fairly frequently will remember that after exploratory celiotomy frequently the fluid would disappear promptly.

DR. DAVID H. WATERMAN (Closing).—I should like to thank the discussants for their remarks and for their approbation. We have been particularly impressed by this group of malignancies that Dr. Kemler and several others have mentioned. Certainly, we would not wish to imply that decortication should always be done in malignant effusion; obviously not. When there is an unexpandable lung with intractable fluid, however, as not infrequently occurs, the operation may be of practical use. Our first case, in 1945, was remarkably successful in eliminating the troublesome effusion, and our subsequent cases have also done well, with the exception of one patient who may have had his death speeded by the opening of new channels for malignant spread, as Dr. Meade mentioned.

It would be presumptuous of me to take issue with an authority on decortication of Dr. Samson's physical and professional stature. I was quoting an article that appeared in 1951 in the Journal of this Association by Croxatto and Sampietro, the former a pathologist and the latter a thoracic surgeon at the University of Buenos Aires, who emphatically stated that there was actually a pachypleuritis on the parietal side and not a true peel, and that it was therefore not possible to separate a peel from the parietal pleura. We had felt that it was possible, and had always used the term "parietal peel" in the past.

We suggest that perhaps a peel is present in the very early cases, and a pachypleuritis in the long-standing ones. However, we do think that removal of the parietal peel or parietal pleura, whichever the case may be, is mandatory in instances where there is marked thickening. It is inconceivable to me that a thick shoe-leather peel over the parietal surface will disappear following decortication of the visceral side alone and permit return of good function of the chest wall.

Dr. Sarot's* comment as to the misnomer perhaps is well taken. I doubt if we are going to be able to change the terminology at this late date because it is so well ingrained in the literature.

I would like to mention briefly one factor that he stressed, namely, the condition of the underlying lung. Obviously it is extremely important to evaluate the pre-existing and existing pulmonary disease if a good result is to be obtained. This is particularly true in tuberculosis. If no appreciable disease is present, however, the duration of collapse does not seem to affect the re-expansion. In our cases of calcified hemothorax, for example, one had been collapsed thirteen years, one nineteen years, and the other twenty-two years since the original gunshot wound. Yet in each case the lung decorticated satisfactorily with the use of bone instruments—the plaque had to be literally chiseled and rongeured away, but once it was removed the lung expanded beautifully, with complete space obliteration on the table.

As a point of interest, the case of calcified hemothorax of twenty-two years' duration had an unsuspected bronchogenic cancer in the upper lobe. This was completely masked by the overlying calcium, and it was not until decortication that the tumor was found. Unfortunately, it was not resectable. Conceivably we have in such instances another rare indication for decortication.

*Dr. Sarot did not return his discussion. For that reason it could not be included here. EDITOR.

A SURGICAL-PATHOLOGIC CLASSIFICATION FOR ISOLATED VENTRICULAR SEPTAL DEFECTS AND FOR THOSE IN FALLOT'S TETRALOGY BASED ON OBSERVATIONS MADE ON 120 PATIENTS DURING REPAIR UNDER DIRECT VISION

HERBERT E. WARDEN, M.D.* (BY INVITATION), RICHARD A. DEWALL, M.D.** (BY INVITATION), MORLEY COHEN, M.D. (BY INVITATION), RICHARD L. VARCO, M.D., AND C. WALTON LILLEHEI, M.D.
MINNEAPOLIS, MINN.

BETWEEN March 26, 1954, and May 5, 1956, at the University of Minnesota Hospitals, 144 patients with intracardiac lesions underwent direct vision repair of their defects while under total cardiac by-pass and were arterially perfused: via controlled cross circulation[1-6] (45 patients); the arterial reservoir[7, 8] (5 patients); the biologic (dog lung) oxygenator[9] (14 patients); or the mechanical (bubble) oxygenator[10, 11] (80 patients).

In this series the most common congenital defect encountered has been an isolated ventricular septal defect (87 patients†). Next in frequency was the tetralogy of Fallot defect (33 patients), and then the lesion of atrioventricularis communis (9 patients). These three types comprise 89.5 per cent of the cases operated upon. The first two types of ventricular septal defects will receive our attention in this publication. The atrioventricularis communis defect, although possessing a ventricular component, has in addition an ostium primum defect and, as well, mitral and/or tricuspid valvular malformations. Therefore, in the light of its complexity, it warrants and will receive a separate communication.

The common denominator in all of these patients operated upon was their recurring illness. They were periodically in need of pediatric or medical care. This fact may well have influenced for this series the frequency distribution of the various defects.‡ The acyanotic cases were in difficulty from numerous respiratory infections and/or bouts of cardiac failure. Pulmonary hypertension was universally present here. Among those who were cyanotic, our surgical indications were based on progressive and sustained deterioration as manifested by one or more of the following conditions: a rising hemoglobin and/or

From the Department of Surgery, and the Variety Club Heart Hospital, University of Minnesota Medical School, Minneapolis, Minn.

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*Research Fellow, United States Public Health Service.

**American Heart Association Research Fellow.

†This includes one case of traumatically acquired ventricular septal defect successfully repaired.[9]

‡The ratio of low septal defects to high ones may be greater than our series shows. The pressing need to care for children in serious trouble from a high ventricular septal defect has effectively precluded scheduling of the more bland forms occasionally met with in older children and adults.

hematocrit (with the emphasis upon the rate at which this ascent was occurring); recurrent severe bouts of dyspnea associated with syncope, coma, or convulsions; and, in some, clearly evident cardiac decompensation.

CLASSIFICATION OF VENTRICULAR SEPTAL DEFECTS

It is both logical and desirable to classify the diverse types of septal anomalies. Consequently, we have sought a simple yet concise and inclusive classification for ventricular septal malformations as we have found them at operation. One was looked for which would be both morphologically correct and equally compatible with the antecedent embryologic aberrations. Rokitansky's anatomic classification of congenital cardiac septal defects as described in 1875,[12] we believe, best fulfills most of the above requirements. We have found that it offers the surgeon an accurate, workable, anatomic classification which, except for a few minor reservations, is essentially in agreement with the embryologic descriptions of Spitzer[13] and others.[14-16] The portion of this classification that deals with ventricular septal defects in its modified form is reproduced in Table I.

TABLE I. ANATOMIC CLASSIFICATION OF VENTRICULAR SEPTAL DEFECTS (AFTER KARL FREIHERR VON ROKITANSKY* 1804-1878)

A. Complete absence of ventricular septum (single ventricle)
B. Defect in the posterior ventricular septum
C. Defect in the anterior ventricular septum
1. Absence of the entire anterior septum
a. With normal caliber of the arterial trunks
b. With stenosis or atresia of the pulmonary artery (tetralogy of Fallot)
2. Defect in the posterior part of the anterior septum
a. With anomalous position of the arterial trunks
Alpha = With normal caliber of the arterial trunks
Beta = With stenosis or atresia of the pulmonary artery (tetralogy of Fallot)
b. With normal position of the arterial trunks (membranous type)
3. Defect in the anterior part of the anterior septum
D. Defect in unusual positions (including muscular defects)
E. Defect in anomalous septum
*F. Acquired ventricular septal defects

*With modifications described in text.

A cross section of the heart at the level of the atrioventricular ring is shown (Fig. 1, *A* and *B*) to facilitate the description and identification of the areas concerned in Rokitansky's classification. The relationship between these structures and the ventricular septum is schematically represented in Figure 1, *C*. The latter is taken from the works of Spitzer[13] and for simplicity has been modified slightly. The stippled areas represent the muscular portions of the ventricular septum while the solid line (*3-5*), joining these regions, represents the pars membranacea. The ventricular septum at this level may be segmented into an anterior or ventral portion (Fig. 1, *C*, *1-4*) and a posterior or dorsal part (*4-6*) which corresponds to the embryologic source of these regions.

The anterior septum is derived from a union of the ventral atrioventricular cushion with the muscular ventricular septum below and the right and left conus ridges from above. The posterior ventricular septum arises from the dorsal atrioventricular cushion and the portion of the muscular ventricular septum that arises from the apical and dorsal ventricular walls of the embryo.[14, 15] The

central point of fusion of these regions occurs at *4* (Fig. 1, *C*) in the pars membranacea. The anlagen of the constituents of this membranous area of the septum have been a subject of dispute among embryologists for some time.

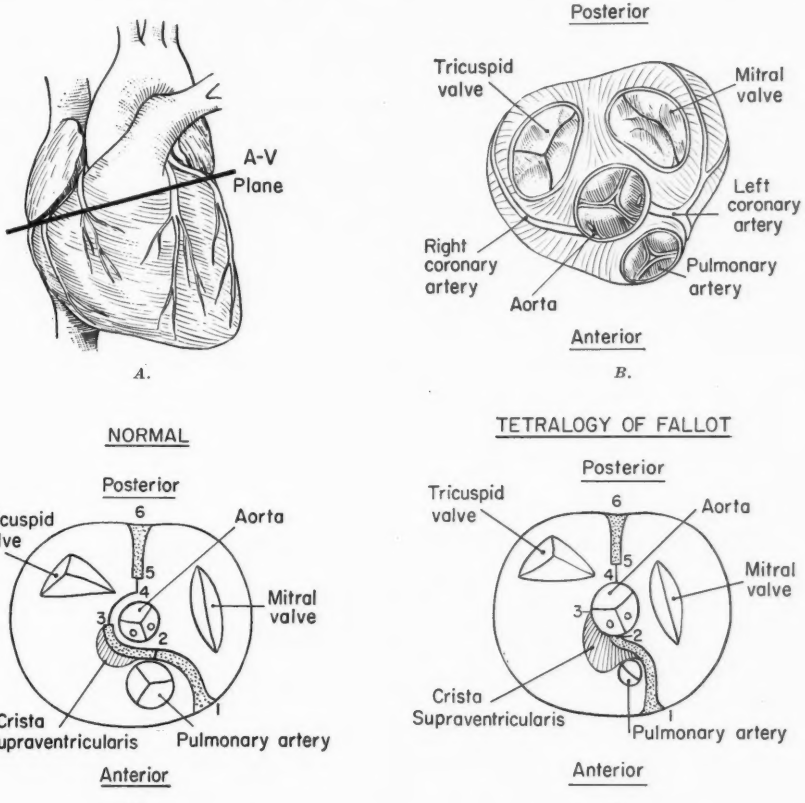


Fig. 1.—The heavy cross bar in *A* indicates the level of the atrial-ventricular junction. *B* depicts the gross anatomy at this level.

The relationships of the mitral and tricuspid valves and the aorta and pulmonary artery with respect to the ventricular septum of the normal heart are schematically indicated in *C*. The stippled areas indicate the muscular portion of the ventricular septum while the heavy line between numbers *3* and *5* indicates the membranous section. In *D*, the pathologic cardiac anatomy found in tetralogy of Fallot is indicated. (See text for details concerning the septal divisions indicated in the latter two plates.)

However, in summary, the present concept holds that the anterior superior portion of the membranous septum arises from connective tissue contributed by the left conus ridge which extends down from the base of the left bulbar ridge while the posterior superior portion comes from the right conus ridge; these segments then join and in turn unite with the fused atrioventricular cushions. The remaining inferior portion of the membranous septum is contributed mainly by the dorsal atrioventricular cushion though some does come from its ventral counterpart and from the connective tissue margin of the crest of the muscular

interventricular septum.[15] Thus, it is obvious that this region has a complex embryologic origin and that fact undoubtedly contributes to the frequency of malformations in this area.

These major divisions of the ventricular septum are further subdivided by Spitzer as indicated in Fig. 1, *C*. The anterior ventricular septum is composed of anterior and posterior divisions. The anterior division extends from the anterior ventricular wall (*1*) to a point midway beneath the aorticopulmonic septum (*2*); the posterior portion (*2-4*) is subdivided into a muscular part (*2-3*) and a membranous section (*3-4*) which extends to the anterior margin of the posterior ventricular septum (*4*). This latter structure is made up of a small membranous segment (*4-5*) and a larger muscular section between *5* and *6* which terminates at the dorsal ventricular wall. These subdivisions by Spitzer, based upon embryologic derivation have been correlated with Rokitansky's morphologic classification and the clinical cases have been grouped according to these terms in Table II.

TABLE II. CLASSIFICATION OF THE CLINICAL MATERIAL IN TERMS OF ROKITANSKY'S GROUPS AND SPITZER'S AREAS

ROKITANSKY'S ANATOMIC GROUPS	SPITZER'S SEGMENTS	NUMBER OF PATIENTS REPAIRED
A	1-6	--
B	4-6	5
C 1 a	1-4	1
b (with pulmonary stenosis)	1-4	3
C 2 a alpha	2-4	2
beta (with pulmonary stenosis)	2-4	30
C 2 b	2-4	71
C 3	1-2	4
D	No correlation	3*
E	No correlation	1
F Traumatic	--	1
Total		121

*One of these also had a C, 2b type defect.

The areas just described and shown in Figure 1, *C* have proved adequate for categorizing the majority of the ventricular septal defects we have observed in this series and those encountered in the tetralogy of Fallot group. The anatomic features of this latter malformation have been diagrammed in Fig. 1, *D* which shows the enlarged aorta, the hypertrophied crista supraventricularis, and the narrowed pulmonary outflow tract. The details of this septal anomaly will be elaborated upon later. The schema here is also modified from the works of Spitzer and the nomenclature is his.

Group A: *Complete Absence of the Ventricular Septum (Single Ventricle).*—

This group is characterized by the complete, or virtually so, failure of the muscular ventricular septum to develop. The atrioventricular valves are at times distorted but empty into a single chamber. In the roof of this common ventricle appear the orifices of the aorta and pulmonary artery. Though these vessels may be normal, frequently they too are imperfectly formed or transposed. Occasionally, the pulmonary artery is markedly atretic or completely absent.

A.

B.

Fig. 2.—Single ventricle (Rokitansky's Group A). These are photographs of a heart from a 4-month-old infant and demonstrate the pathologic anatomy of one type of single ventricle with normal position and caliber of the great vessels. *A* shows the pulmonary outflow tract in the upper right foreground. *B* is a view from the apex toward the roots of the ventricular outflow tracts. In each instance the probe extends out the aorta.

The aorta can be similarly deformed. Fig. 2 (*A* and *B*) are photographs of a representative specimen of one type of single ventricle with normal atrioventricular valves and normal position and caliber of the great vessels. In Fig. 2, *A* the incision in the ventricular cavity has been extended from the apex well out the pulmonary artery and the orifice of the latter structure is in the foreground. A probe passes out through the aorta. In Fig. 2, *B* the specimen has been more widely opened and the base of the great vessels is more apparent as are the tricuspid and mitral valves. The probe again extends out the aorta. As yet we have not undertaken surgical correction for this group of cases.

Group B: *Defect in the Posterior Ventricular Septum.—*

Defects of this portion of the ventricular septum are represented in Fig. 3 (*A* and *B*). It can be noted that here one margin of this defect is in juxtaposition to the atrioventricular ring or annulus, and that it is well concealed by the septal and, to some extent, the posterior leaflets of the tricuspid valve. In addition, because of a close proximity to the atrioventricular ring, the upper margin of such a defect is usually quite fibrous while the lower margin is composed predominantly of muscle.

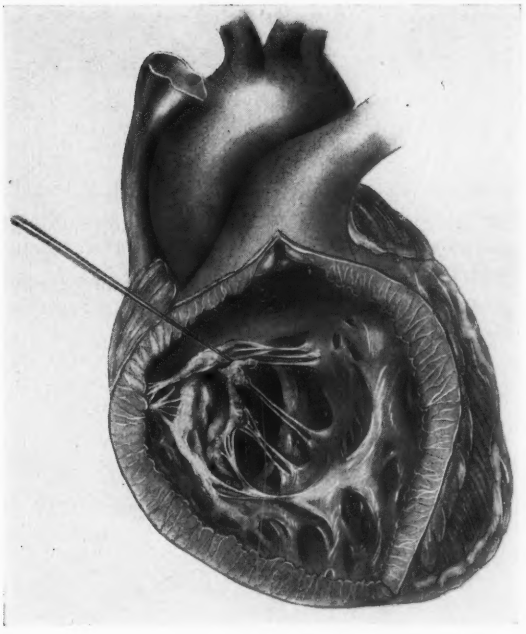
These lesions may vary from that of a hole but a few millimeters in diameter and juxtaposed to the posterior margin of the membranous septum to an almost total absence of the posterior septum extending from the pars membranacea to the posterior ventricular wall. We have cataloged 5 patients in this series as having this type of defect. In 3 cases the opening appeared smaller than that shown in Fig. 3, *B*. Their range was between 1.5 and 2.5 cm. However, among these smaller holes encountered, adequate surgical exposure may be more troublesome and hence secure closure more difficult.

Group C: *Defect of the Anterior Ventricular Septum.—*

1. *Absence of the entire anterior septum:* A heart with this portion of the septum deficient and with normal caliber of the arterial trunks is illustrated in Fig. 4. The openings found so far have all been large and extended from just beneath the pulmonary valve cusps well back toward the anterior margin of the septal leaflet of the tricuspid valve. The aortic and pulmonary valve leaflets are separated by a narrow (2 to 4 mm.) fibrous band, leaving just enough room for the precise placement of closure stitches.

Four patients in the series had this lesion. In only one was there a normal outflow tract. The others had definite pulmonary outflow stenosis (tetralogy of Fallot). This is an unusual form of tetralogy since the supraventricular crest has appeared to be entirely absent, whereas, it is customarily hypertrophied (C, 2a, beta). The appearance at the time of right ventriculotomy is strikingly different in these two contrasting examples. Rokitansky seemingly did not observe any defects of this portion of the ventricular septum in association with pulmonary stenosis or atresia and hence did not make provision for their inclusion in his classification. Consequently, we have modified his original schema (see Table I) by this addition.

Group C, 2a, alpha: *Defect in the posterior portion of the anterior septum with anomalous position of the arterial trunks which are of normal caliber:* This group includes those septal defects which are associated with anomalies



A.

B.

Fig. 3.—Defect of the posterior septum (Rokitansky's Group B). *A* is an artist's version of a defect in the posterior ventricular septum and *B* shows a photograph of a similar defect taken from a view similar to that seen through a surgical cardiotomy in the right ventricle. In this view the pulmonary valve cusps are visible at the top of the photograph and the manner in which the septal and posterior leaflets of the tricuspid valve tend to hide partially the margins of the defect is quite evident.

of the position or origin of the great vessels. These malpositions range all the way from partial to complete transposition of the great vessels. The septal defect is usually located in or near the membranous area but, of course, may occur elsewhere as well. Certainly, because of the malposition and rotation of the great vessels, considerable variation is induced in the site of the septal defect. Fig. 5 is a representative specimen of the group. This photograph shows complete transposition of the great vessels and a ventricular septal defect. The right atrioventricular valve is apparent in the left foreground and just above its apex appears the septal defect. In the upper center of the field may be seen the aorta as it exits from this anterior ventricle.

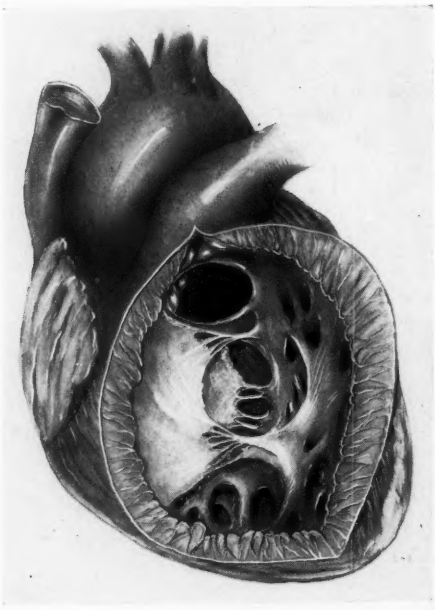


Fig. 4.—Absence of the entire anterior septum (Rokitansky's Group C, 1). This type of anterior septal defect with normal caliber of the arterial trunks is diagrammatically shown in this plate. Note the proximity of the aortic and pulmonary leaflets.

A discussion of this group is included primarily for completeness of the classification. Two patients with this type of ventricular septal defect have been operated upon as part of an attempt at complete corrective surgery for complete transposition of the great vessels.[17]

Group C, 2a, beta: *Defect in the posterior part of the anterior septum with anomalous position of the arterial trunks and stenosis or atresia of the pulmonary artery (tetralogy of Fallot):* Under this heading, Rokitansky has grouped those defects associated with an enlarged, dextraposed aorta and stenosis or atresia of the pulmonary artery. Thirteen years later, Fallot[18] also described the pathologic anatomy of these defects and correlated it with the clinical findings in patients possessing that combination of congenital intracardiac

anomalies. Since his classic description in 1888 this tetralogical association has attained common usage whenever reference is made to that pathologic complex.

The significant abnormalities in this syndrome are the ventricular septal defect and stenosis or atresia of the pulmonary artery. The positional alterations in the aorta and pulmonary artery come in part from a faulty partitioning of the truncus arteriosus by the septum aorticum,[12, 13] and in part are acquired secondarily, after birth, from the disproportionate blood flow through these respective vessels. The result is an inequality in the size of these two vessels which, together with certain other factors,[5] contributes to the impression that the aorta is overriding. Thereafter, the aorta encroaches upon this area normally occupied by the posterior portion of the anterior septum (see Fig. 1, *D, 2-4*). Although the pulmonary artery is always the smaller of the two vessels its actual size may vary from an atretic cord or hypoplastic vessel to that of an artery scarcely diminutive to the normal.

Fig. 5.—Defect in posterior part of anterior septum with anomalous position of the arterial trunks (Rokitansky's Group C, 2a, alpha). An example of the defects included in this group is the above photograph from a 4-month-old child dying of a complete transposition of the great vessels. The aorta, located anterior to the pulmonary artery, can be seen coming off the right (anterior) ventricle. The defect involving the posterior part of the anterior septum is seen in the center just above the commissure of the right atrioventricular (tricuspid) valve.

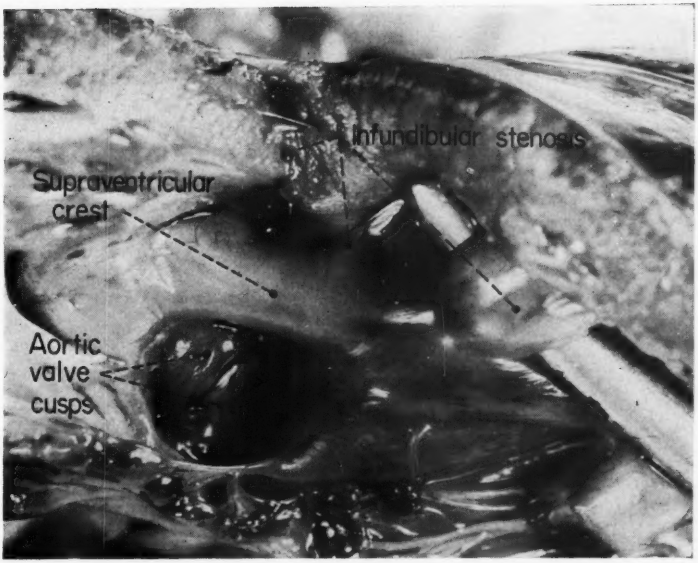
Associated with a slight malposition of the aorta, there is also an exaggerated prominence of the crista supraventricularis. The latter, quite like the pulmonary artery, may exhibit considerable variation in size. It can be completely absent, as described in Group C, 1b, or it may exist as a moderately thickened muscular ridge. In its extremely overdeveloped form, it appears as a markedly hypertrophied muscular mass obstructing the right ventricular outflow tract to a pinhole. In the latter instance, it usually forms the floor of a rather well-developed infundibular chamber proximal to the orifice of the

pulmonary artery. At times the encroachment through hypertrophy is capable of completely obliterating this region. Finally, through an asymmetrical division of the truncus arteriosus, there is frequently a deficiency in the number of valve cusps, resulting not uncommonly in either a bicuspid aortic or pulmonic valve.

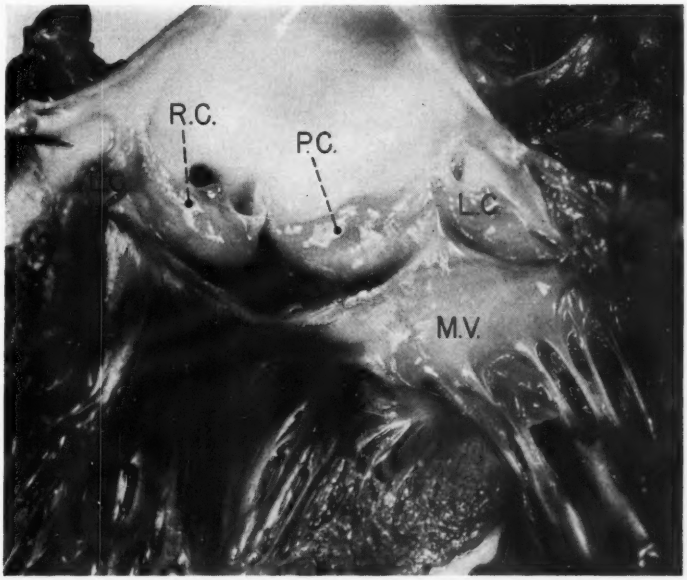
The septal defects noted in this group are generally located in the membranous area of the septum and have been generally large in size. However, despite the similarity in location they differ considerably in appearance from the pure membranous type of defect. Characteristically, they show an extremely narrow upper margin immediately adjacent to the origin of the aortic cusps from the fibrous base of the aortic ring (Fig. 6, *B*). The remaining margin is predominently muscular as a rule but occasionally may consist too of a rather narrow shelf of fibrous tissue. This close proximity of the aortic cusps is seen in Fig. 6, *A*, an autopsy specimen with the view from the right ventricular side. In the lower left-hand corner lie the tricuspid valve orifice and leaflets while just above and medial to this is the septal defect through which appear two of the aortic cusps. This patient died before corrective surgery could be performed and therefore the thickened crista supraventricularis is shown as an intact ridge which traverses the right ventricular outflow tract. In this field on the right of the crista is a broad band of muscle extending from the septum to the anterior ventricular wall. The tip of a hemostat passes beneath this structure. This configuration has been found in the majority of cases in this group. Unquestionably, it serves to obstruct blood flow into the pulmonary artery. We have excised it therefore since its removal augments substantially the volume of blood moving through the outflow tract. Fig. 6, *B* depicts the septal defect as seen from the left ventricular aspect. Again, the proximity of the right and posterior aortic valve cusps to the typical crescent shape of the defect is apparent.

To date 33 patients with tetralogy of Fallot have been operated upon. In 29, the location of the septal defect was of this type (C, 2a, beta). Three of the other cases had deficiencies of the entire anterior septum as previously noted; the fourth patient, who preoperatively was thought to have a tetralogy of Fallot, had an atrioventricularis communis defect of the septum with deformed mitral leaflets and a severe pulmonary valvar stenosis. One patient with tetralogy of Fallot in this series had complete atresia of the origin of the main pulmonary artery (see Fig. 11, *A*). She continues to do well one and one-half years postoperatively after correction of that anomaly also.[5] In this group, just one patient with tetralogy of Fallot had only valvular stenosis. Twenty-seven had infundibular stenosis and 5 had both types of obstruction to pulmonary outflow. Three patients had other congenital cardiovascular abnormalities; one had an associated patent ductus arteriosus which was divided, one had a left superior vena cava entering the coronary sinus, and one also had a primum defect of the interatrial septum. Right aortic arch was present in approximately one-third of these patients.

It is important to emphasize, for surgeons and cardiologists alike, the fact that the position of the aorta has not presented any important obstacle to curative surgery for Fallot's tetralogy, even in its severest forms encountered in infancy.

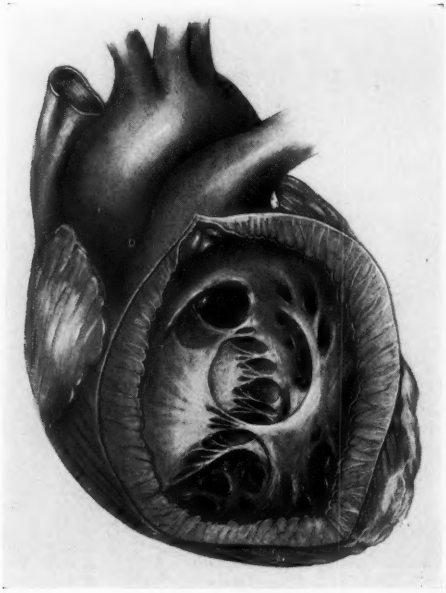


A.



B.

Fig. 6.—Defect in posterior part of anterior septum with anomalous position of the arterial trunks (Rokitansky's Group C, 2a, beta). This is an example of a tetralogy of Fallot in a 2-month-old infant. *A* is a close-up view from within the right ventricle looking toward the aortic and pulmonic outflow tracts. In the upper portion is seen the markedly stenotic pulmonary outflow tract and to the right a large muscular trabeculae (tented up by the probe) which frequently has been found obstructing the outflow tract in this region and should be excised at the time of corrective surgery. In the lower central portion are seen the aortic cusps through the high interventricular defect and in *B* the wide crescent-shaped defect is seen from the left ventricular side and demonstrates the close proximity of the aortic valves to the septal defect. The aorta is seen arising entirely from the left ventricle. The right aortic cusp (R.C.) and the posterior cusp (P.C.) of the aortic valve are shown.

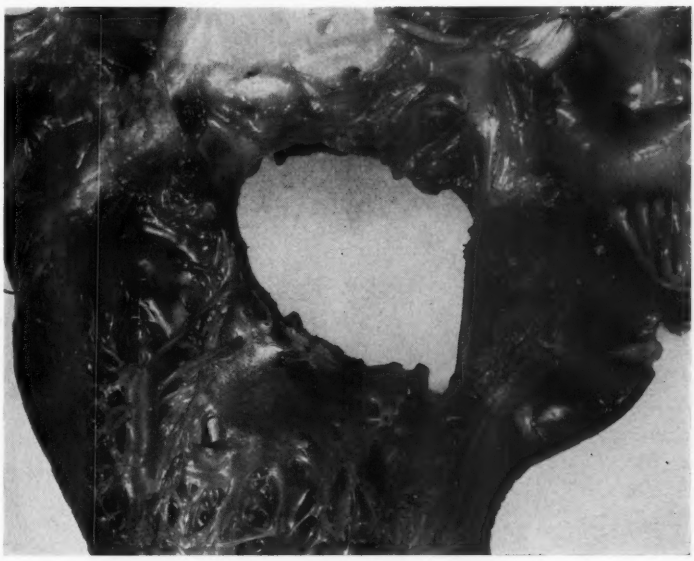


A.

B.

C.

Fig. 7.—Defects in the membranous septum with normal position of the arterial trunks (Rokitansky's Group C, 2b). *A* demonstrates diagrammatically the typical membranous defect. Note how the defect margin extends beneath and is partially hidden by the tricuspid leaflets. *B* is a right ventricular photograph of such a defect. Note the relationship of this defect to the crista supra-ventricularis (C.S.) and the papillary muscle of the conus (P.M.C.). *C* is a view of the same defect viewed from the left side. The relationship of this type defect to the right aortic cusp (R.C.) and the posterior (non-coronary) cusp of the aortic valve is evident.



D.

E.

Fig. 7 (Cont'd).—*D* indicates a defect of this type viewed from the left side, occurring in a 4-year-old boy. Defects in this area grow disproportionately larger with time. *E* shows a somewhat unusual type of membranous septal defect found in a 5-week-old infant. This is viewed from the right ventricular side and in the central portion shows an aneurysmal dilatation of the membranous septum and a perforation through the septal leaflet of the tricuspid valve, thus creating a direct communication between the left ventricle and the right auricle. (See text for details.)

Group C, 2b: *Defect in the posterior part of the anterior septum with normal position of the arterial trunks (defects of the membranous septum):* A defect in the membranous portion of the ventricular septum with normal location of the aorta and pulmonary artery was the most frequent congenital intracardiac malformation encountered in this series. These 71 abnormal ostia varied in diameter from 5 mm. to 6 cm. (Fig. 7, *D*) depending upon the amount of adjacent muscular septum that also was deficient.

As sketched in Fig. 7, *A*, the posterior margin can be difficult to expose for it lies behind the papillary muscle of the conus, concealed by the anterior commissure of the tricuspid valve. In reality no true defect margin need exist in this area. The edge of the septal hiatus merges with the annulus fibrosus of the tricuspid and mitral valves and this in turn diffuses into the aortic septum. Surgical exposure is also limited by attachments of the chordae tendineae to the inferior margin of the septal defect along its mid to anterior position. Occasionally, these chordae pass through the defect and insert on the left ventricular side of the septum. The usual characteristics of this lesion are shown in Figs. 7, *B, C, D.* Plates *B* and *C* are photographs of a specimen* viewed from the right and left ventricles, respectively. The septal hole is centrally located under the normally thin, broad, shelflike projection of the crista supraventricularis which here has an entirely different configuration from its appearance in a tetralogy of Fallot and hence it does not infringe on the pulmonary outflow tract. Fig. 7, *C,* a photograph of the same specimen, seen from the left ventricle, demonstrates the relationship of the aortic valve to this membranous septal defect. Characteristically, in this type of defect, the aortic cusps are located a few millimeters farther away from the upper edge of the hole than when this kind of anomaly exists in a tetralogy. In the latter, this edge is usually very close to the aortic cusps. These relationships are of considerable importance in the placement of sutures during the repair.

Of the seventy-one membranous septal defects seen in this series of patients, four have had a definitely different appearance. The ventricular defect in each instance was in the usual location but arose from an aneurysmal dilatation of, and perforation in, the membranous septum. One infant had in addition a perforation through the origin of the septal leaflet of the tricuspid valve. This resulted in a direct shunt from the left ventricle into the right auricle. A defect similar to this has been described by Rokitansky,[12] and Edwards and associates.[19] The specimen from this case is shown in Fig. 7, *E*. The heart was opened so as to expose the right auricular and ventricular cavities. To the right of center lies the aneurysmal pouch and within its apex are two oval defects through which can be seen the margins of the membranous septal defect in the background. This unusual lesion produced an intolerable hemodynamic shunt. This infant though merely 6 weeks old was moribund when operated upon as a last resort.

*This patient, J. D., age 16 weeks, died in the hospital while awaiting his operation.

Twenty-six patients in this group had other cardiovascular anomalies. Among these there were five instances of left superior venae cavae. Four patients had a patent ductus arteriosus which was transected at the time the other corrective surgery was performed. Three had associated atrial septal defects which were likewise repaired. Mitral stenosis was found in one patient with a defect in the membranous ventricular septum. He underwent commissurotomy under direct vision at the time the ventricular septal defect was closed. The remaining 2 patients had coaretation of the aorta.

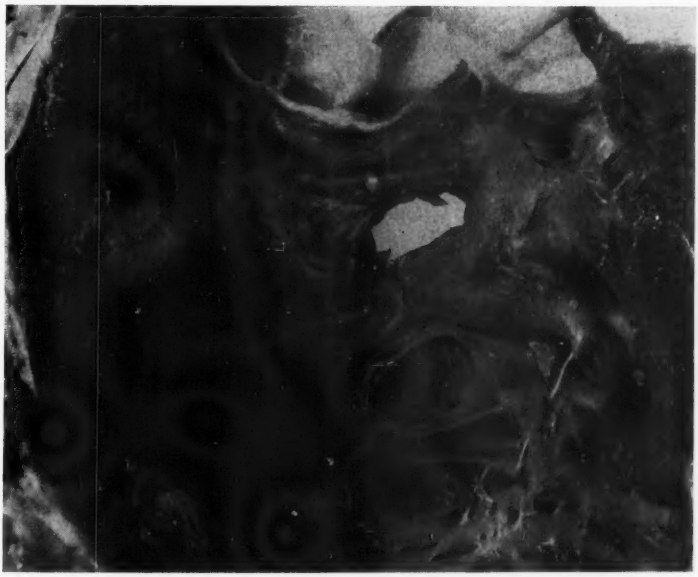
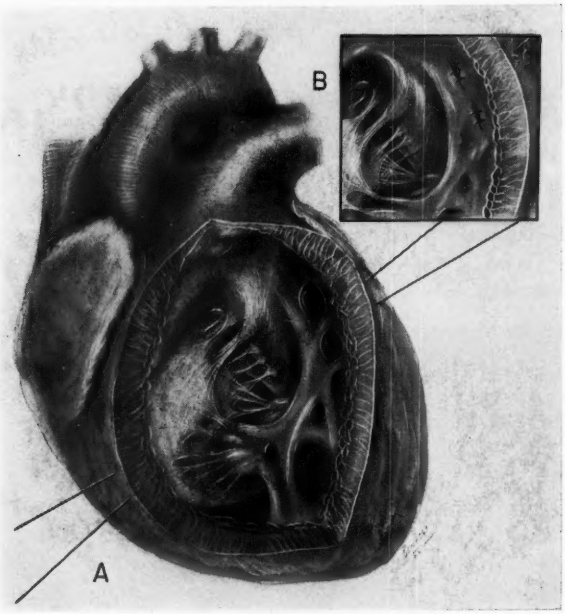


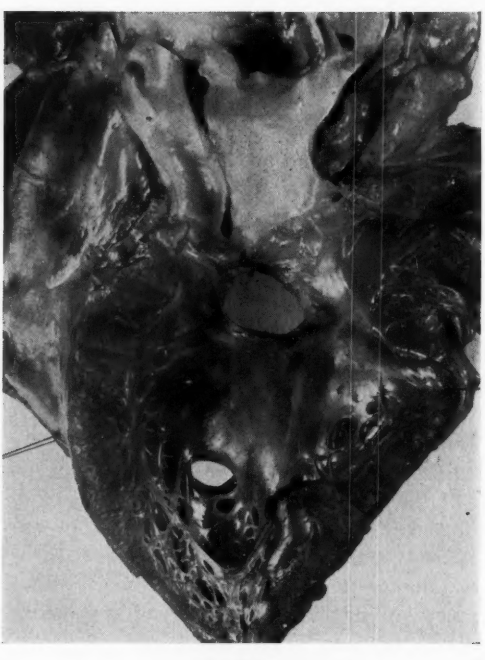
Fig. 8.—Defect in the anterior part of the anterior septum (Rokitansky's Group C, 3). Note the proximity to the pulmonary valves.

Group C, 3: *Defect in the anterior part of the anterior septum:* Defects of the muscular portion of the anterior ventricular septum occur in the area between *1* and *2* in Fig. 1, *C*. This type defect is usually located close to the anterior ventricular wall just below the pulmonary valves (Fig. 8). Those we have found were 4 to 15 mm. in diameter. Uniformly, they have had a muscular margin inferiorly. In this defect, similar to that mentioned above in regard to absence of the entire anterior septum, the aortic valve (left anterior cusp) and the pulmonary valve leaflets are separated by only a narrow (2 to 4 mm.) margin. Hemodynamically also, they unite the outflow tracts of the right and left ventricles (similar to Group C, 1) and hence provoke severe systemic effects. The four met with in this series were readily visualized and the patients tolerated the surgical closure well.

Group D: *Defect in Unusual Positions (Including Muscular Defects).*—Three patients have been found at operation to have this type of deficiency of the muscular portion of the ventricular septum. Two had associated cardiac



A.



B.

Fig. 9.—Defects in unusual locations (including muscular defects) (Rokitansky's Group D). *A*, multiple muscular defects occurring as the sole defects in a seriously ill 5-month-old infant. Successfully repaired in 1954, utilizing controlled cross circulation. *B*, specimen from a 6-year-old boy which shows a moderate-sized muscular defect low in the septum in addition to a large membranous septal defect as seen from the left ventricular side.

lesions. One had a large, yet correctable and typically located, defect in the membranous area as well. The other patient had mitral stenosis too, which was relieved under direct vision after closure of the ventricular defect. In all, the defects had friable muscular margins and their diameters ranged from 5 to 15 mm. The third patient had multiple muscular defects (Fig. 9, *A*). Frequently several trabeculae carneae overlay the right ventricular orifice of the defect thus creating an initial impression that a cluster of several small defects exists. However, once these trabeculae are incised it is apparent that these ostia coalesce into a single opening perforating the septum. Occasionally, as we have noted, the holes may truly be multiple (Fig. 9, *A*). The more common sites are along the muscular septum at almost any level subjacent to the anterior ventricular wall but on occasion we have found them along the posterior ventricular wall. When present in the central portion of the muscular septum they have been near the apex. The specimen seen in Fig. 9, *B* demonstrates this situation as seen from the left ventricle. Here, the low muscular septal defect is associated with a rather large membranous opening as well. That particular combination occurred once in the operative series and both defects were successfully closed.

Group E: *Defect in Anomalous Septa.*—

Rokitansky has included under this heading a multiplicity of defects involving malposed septa, for example, those associated with a rudimentary ventricle and various gradations of transposition as well as the situation in which both great vessels come from either the large or small ventricle in combination with varying degrees of anomalous venous inflow to the heart. We have recognized only one patient in this category among our operative series, but have seen other examples at autopsies. Our patient had a "corrected" transposition by virtue of the anomalous septum together with large ventricular and atrial (secundum) defects.

Group F: *Acquired Ventricular Septal Defects.*—

Acquired ventricular septal defects can develop after a crushing chest injury, bacterial endocarditis, or from coronary infarctions. We have had an operative experience with the former.[9] The patient sustained multiple rupture sites in the ventricular septum following compression of the thorax under a rolling truck wheel. The three defects were located in the muscular septum near the apex of the heart. They possessed heavily scarred margins, were easily approximated by silk stitches, and the patient remains well one and one-half years, postoperative. This group is an addition to Rokitansky's classification that seemed indicated, for completeness, in light of clinical experience.

ASSOCIATED DEFECTS

Among this group of patients who underwent direct surgical repair of their ventricular septal defects, a number were found to have other associated cardiovascular malformations. These have been recorded in Table III. As a result of this experiment we have come to believe that it is probably wise to correct the coexisting anomaly either primarily or coincidentally with repair of the ventricular defect. This, of course, is not applicable to endocardial fibroelastosis,

TABLE III. ASSOCIATED CARDIAC MALFORMATIONS ENCOUNTERED DURING REPARATIVE SURGERY VENTRICULAR SEPTAL DEFECTS INCLUDING TETRALOGY OF FALLOT

PRIMARY DEFECT	NUMBER PATIENTS	MULTIPLE VSD	ATRIAL SEPTAL DEFECT	PATENT DUCTUS ARTERIOSUS	COARCTATION	ENDOCARDIAL FIBROELASTOSIS	CORRECTED TRANSPOSITION	LEFT SUPERIOR VENA CAVA	DEXTRA-CARDIAC
Ventricular septal defect	87	3	3	4	2	7	1	5	1
Tetralogy of Fallot	33	0	1*	1	0	1	0	1	0
Total	120	3	4	5	2	8	1	6	1

*Ostinum primum.

corrected transposition, dextracardia, or left superior vena cava. However, it is especially important for the surgeon to recognize the presence of this latter abnormality for unless it is occluded during the by-pass period the operative site is often unnecessarily obscured by this venous return. Also, particularly if this accessory superior vena cava is of any significant size, it is better not to occlude it but rather to cannulate it in order to minimize possibility of cerebral congestion if cross connections are lacking.

Similarly, aside from the physiologic undesirability of leaving an unrecognized coaretation of the aorta or patent ductus arteriosus uncorrected, overlooking the latter lesion at the time of corrective intracardiac surgery may result in catastrophic hemorrhage when the heart is opened.

SURGICAL MANAGEMENT

Spatial limitations require that a detailed discussion of this important consideration be deferred. Currently we find three methods useful. These are: (1) direct suture, (2) Ivalon pledget, and (3) circumferential patch. These types of closure are portrayed diagrammatically in Fig. 10. The particular anatomic configuration found, when the heart is open, decides the choice. The frequency with which each type was used has been summarized in Table IV. The distribution of methods used is somewhat askew, however, because initially many defects were closed by direct suture whereas now we would probably favor the use of an Ivalon pledget or a circumferential patch for many of these cases.

TABLE IV. METHOD OF REPAIR OF VENTRICULAR DEFECTS

TYPE OF DEFECT	NUMBER PATIENTS	STITCHES ONLY	PLEDGET	PATCH
B	5	2	3	0
C 1 a. (without pulmonary stenosis)	1	1		
b. (with pulmonary stenosis)	3	1	0	2
C 2 a. (Alpha)	2	1	1	0
(Beta)	30	10	17	3
C 2 b.	71	37	32	2
C 3	4	1	3	0
D	3*	2	1	0
E	1	0	1	0
F Traumatic VSD	1	1	0	0
Total	121	56	58	7

*One of these also had a C, 2b type defect.

Right Ventricular Outflow Tract Roof for Patients With Tetralogy of Fallot.—

The curative procedure for tetralogy of Fallot has completely replaced the palliative shunt procedure in this clinic. The achievement of this more desirable treatment for these patients involves two considerations; namely, closure of the ventricular septal defect and removal of the pulmonary stenosis. The anatomy and techniques of closure of their ventricular septal defects have been discussed above.

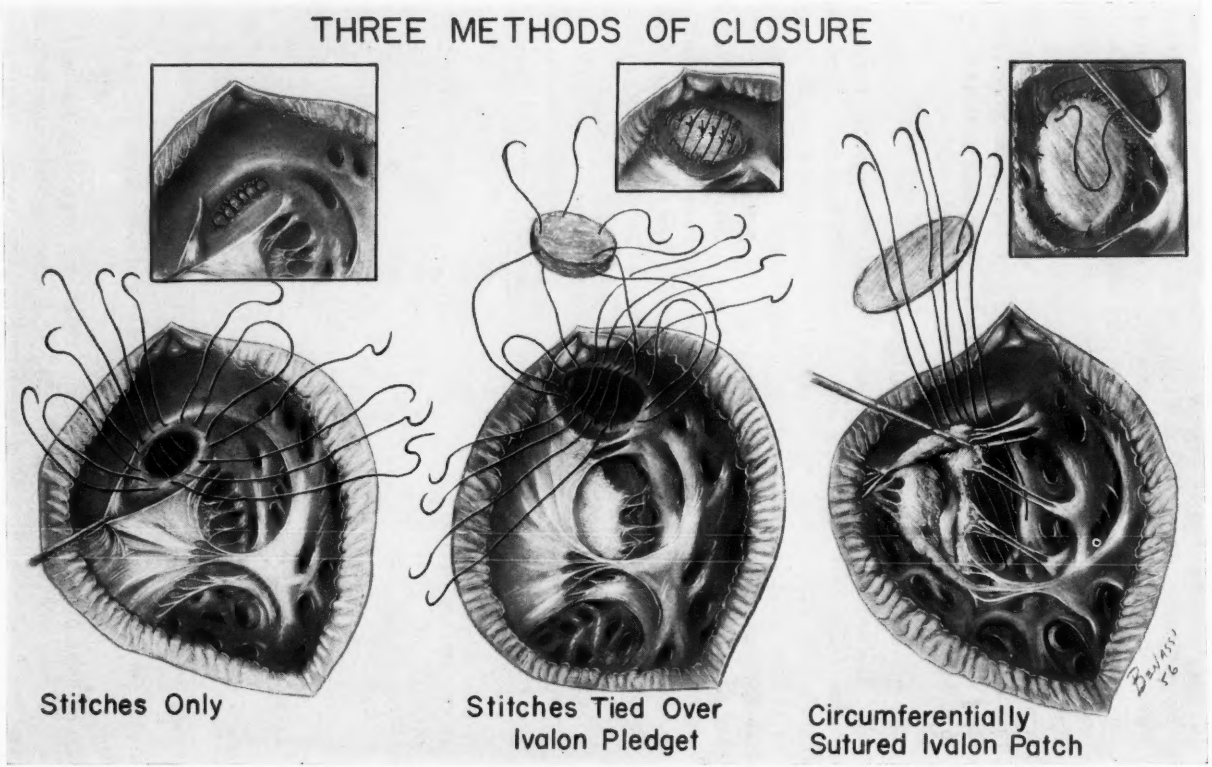


Fig. 10.—The methods of closure of ventricular septal defects. Silk (3-0) with a double-armed swedged-on needle was the suture material utilized in all instances.

Pulmonary valvulotomy and/or infundibulectomy (Fig. 11, *A*, and *B*) has been satisfactory for many of these patients operated upon,[5, 20] but one of the important technical innovations has been the use of a teardrop-shaped piece of compressed Ivalon sutured into the upper portion of the right ventricuiotomy

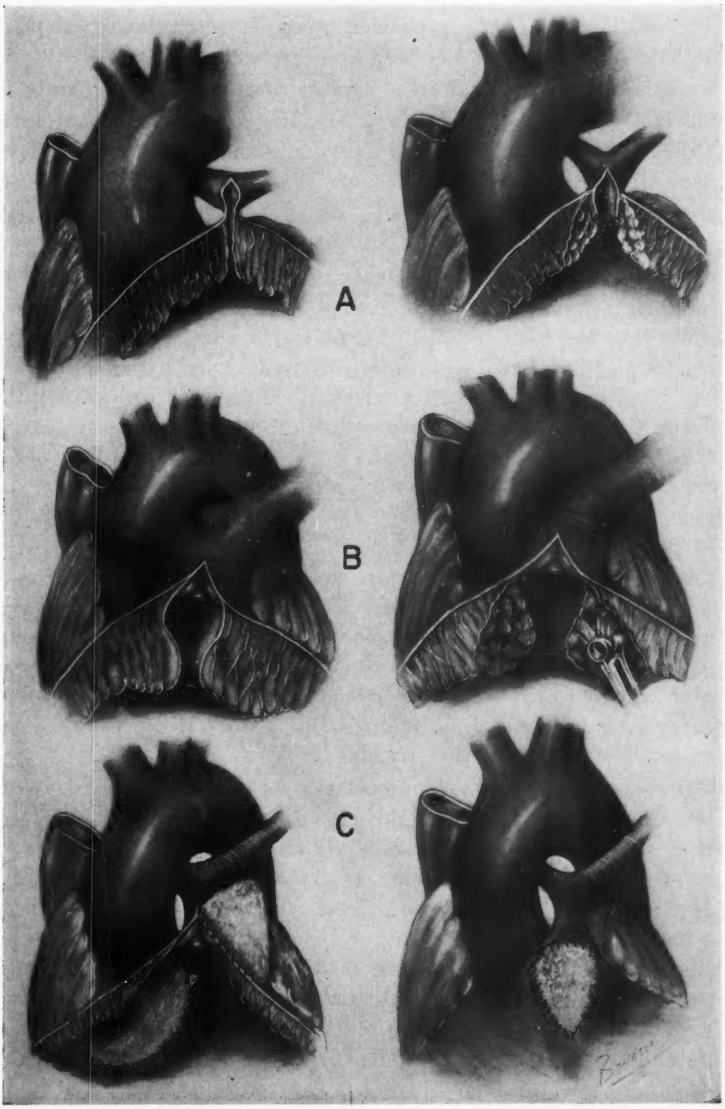


Fig. 11.—Methods of treatment of the right ventricular outflow obstruction in tetralogy of Fallot. *A*, The atretic segment is resected and the pulmonary artery just proximal to its bifurcation is anastomosed to the right ventricle. *B* depicts the technique of infundibulectomy. *C* shows an Ivalon roof used to expand the right ventricular outflow tract. (See text for details.)

(Fig. 11, *C*) to enlarge further this outflow tract in the severe tetrads where it is not anatomically possible to excise a sufficient amount of muscle to accomplish this goal.

DISCUSSION

Our identification of the particular types of ventricular septal pathology seen in this series of cases has been greatly facilitated both by the use of Rokitansky's classification and from familiarizing ourselves with these defects in terms of their embryologic development. His classification has required but few modifications in order to provide a comprehensive coverage, yet still remain a conveniently workable method.

We recognize that the incidence shown in Table II for these various types of defects is unlikely to be a precise reflection of their natural occurrence rate. As indicated previously, the patients in this series were selected on the basis of their deteriorating clinical course. All persons operated upon were, or at some time had been, severely ill and none was asymptomatic. Except for those in tetralogy of Fallot groups, all had pulmonary hypertension. In the majority of them the pulmonary artery systolic pressure was greater than 70 per cent of the aortic systolic pressure. Many suffered repeatedly from severe respiratory infections and those few who were chosen, *because* of their peripheral arterial desaturation, emphasize their desperate need for corrective surgery. The compelling operative indications among the tetralogy of Fallot patients have already been cited.

The site of the most common septal defect encountered in this series was in the pars membranacea. Too, the clear-cut preponderance in frequency of the membranous defects over the other forms of ventricular septal defects substantiates our belief that these lesions are invariably detrimental to the patient. Undoubtedly, from their location a strong pulsatile thrust is imparted to the pulmonary artery and as a consequence severe proliferative changes frequently develop in the arterioles of this vascular bed at an early age.

While there has been some correlation between the size of the defect and the degree of clinical disability in patients with isolated ventricular defects, a factor, at least as important in this regard, has been the precise anatomical relationship of the defect to the orifice of the pulmonary artery. Thus, even relatively small defects (whether they be in the pars membranacea, in the anterior, or in the posterior ventricular septum), situated so that the pulsatile thrust of the bolus of the blood from the left ventricle projects directly into the pulmonary artery, are hemodynamically more deleterious than considerably larger defects situated so that the left ventricular jet strikes the opposite ventricular (right) wall in such a way as to create turbulence in the outflow tract of this ventricle. The consequent increased resistance due to this turbulence creates, in effect, a functional pulmonic stenosis tending to dampen the physiologic consequences of the defect.

In view of the fact that corrective surgery is now possible and since it is an historical fact that these patients with ventricular septal defects rather uniformly are seriously ill and have a severely contracted life expectancy, it would therefore seem pertinent currently to balance for each patient the increasing hazard of procrastination against the decreasing operative risk.

We wish to express our appreciation to Dr. Bernard Hoelscher of the Department of Surgery, University of Minnesota, for his aid in the translation of the original papers of Rokitansky and Spitzer.

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Discussion

DR. DENTON A. COOLEY, Houston, Texas.—I rise to congratulate Drs. Warden, Lillehei, and their associates on this outstanding contribution. The pathologic anatomy of ventricular septal defects must vary more widely than had been previously appreciated. Recently we encountered at operation a defect of the membranous portion of the ventricular septum which resulted in a communication between the left ventricle and the right atrium without entering the right ventricle at all.

Another patient upon whom we recently operated was 49 years old and about ten weeks previously had suffered an extensive posteroseptal myocardial infarction. About one week later, spontaneous perforation of the ventricular septum occurred. At open cardiotomy, using the cardiac by-pass, a defect about 3 cm. in diameter was discovered in the posterior muscular portion of the septum, partly hidden by the septal leaflet of the tricuspid valve. In order to expose the defect, two papillary muscles of the septal leaflet were divided and the defect was closed with interrupted silk sutures incorporating a polyvinyl sponge. This operation was done six weeks ago and the patient is still alive.

One may question the efficacy of such a procedure on the basis that healing would be retarded by the surrounding infarcted muscle. Actually the collateral blood supply to the ventricular septum is good and healing should be satisfactory. The rim of tissue around the defect in this case was quite firm, and I think that a direct suture without the sponge would have been satisfactory. I believe that, in view of the grave prognosis of acquired septal defect, surgical repair of the lesion should always be attempted if the patient's general condition will permit operation.

DR. HERBERT E. WARDEN (Closing).—I should like to thank Dr. Cooley for his remarks. I was afraid of the time limit, consequently I omitted from the presentation two slides which I believe demonstrate the type of anomaly he alluded to. This type has been described in Rokitansky's[12] works on septal defects in 1875 and also by Edwards and associates.[19]

(slide) (See Fig. 7, *E* in text.) This is a photograph of a heart, viewed from the right ventricular side, which shows such a defect. Here, in the center of the photograph, you will note the aneurysmal dilatation of the membranous septum has two perforations adjacent to its apex. In addition, you will note that these two perforations communicate with similar sized perforations in the septal leaflet of the tricuspid valve. The location of these defects, together with the physiologic data obtained at cardiac catheterization, indicated that during life there was a considerable shunt directly from the left ventricle into the right auricle.

(slide) This second slide is a photograph of the same heart viewed from the left ventricular side. Here will be noted the rather typical location of the membranous defect and through it can be seen the concavity of the aneurysmal dilatation of the membranous septum with the openings at its apex.

This is an autopsy specimen from a 4-week-old infant who died in the early postoperative period. This patient was in extremely poor clinical condition preoperatively which can be attributed to this very unusual type of defect. Thus far in this series of patients this is the only one of this type of defect we have encountered. However, 3 other patients with intact tricuspid leaflets, but with a similar type ventricular defect, have been seen.

SURGICAL CORRECTION OF VENTRICULAR SEPTAL DEFECT: ANATOMIC AND TECHNICAL CONSIDERATIONS

JOHN W. KIRKLIN, M.D.,* HARRY G. HARSHBARGER, M.D.** (BY INVITATION), DAVID E. DONALD, B.V.S., M.R.C.V.S.*** (BY INVITATION), AND JESSE E. EDWARDS, M.D.**** (BY INVITATION)
ROCHESTER, MINN.

ANALYSIS and solution of the technical problems inherent in surgical closure of ventricular septal defects must be of concern to all engaged in corrective intracardiac surgery. Knowledge of the anatomy of the lesion and the methods of repair is necessary no matter what technique is used to permit the surgeon to work within the open heart. A study of these problems, initiated in the dissecting room and completed in the operating theater, yielded the results presently reported.

Extracorporeal circulation, utilizing a mechanical pump-oxygenator with a Gibbon-type stationary vertical screen oxygenator, was employed in the cases herein reported. A series of 81 patients operated on by this method has given evidence as to the soundness of the technique. The details of the pump-oxygenator,[1] the methods for cannulation and perfusion during operation,[2, 3] physiologic studies,[4] and mortality rates[4, 5] have been previously reported and will not be repeated herein.

PATHOLOGIC ANATOMY

The concept of the ventricular septum and its defects developed in a previous report[6] from this institution is a logical beginning to an analysis of the challenge posed by repair of ventricular septal defects. The present route of surgical approach being through the right ventricle, the relations as seen from this aspect are particularly pertinent.

Ventricular septal defects may involve regions of the ventricular septum related to the ventricular outflow tracts or regions related to the inflow tracts. The outflow portion of the right ventricle, as defined in the study mentioned, lies between the pulmonary valve above and the nearest portion of the tricuspid valve below. The inflow portion lies posterior and caudal to the outflow tract. Major anatomic landmarks of the interior of the right ventricle are: (1) the tricuspid valve ring and leaflets, (2) the papillary muscle of the conus, (3) the crista supraventricularis, (4) the annulus fibrosus of the pulmonary valve, and (5) the apex of the ventricular septum (Fig. 1).

The papillary muscle of the conus is that point of the septal wall of the right ventricle into which insert the chordae tendineae from the left half of the

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The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

*Section of Surgery, Mayo Clinic and Mayo Foundation.
**Fellow in Surgery, Mayo Clinic and Mayo Foundation.
***Research Associate, Mayo Clinic and Mayo Foundation.
****Section of Pathologic Anatomy, Mayo Clinic and Mayo Foundation.

anterior tricuspid leaflet and the anterior half of the septal leaflet of this valve. In some hearts, particularly those in which the right ventricular chamber is dilated, the chordae tendineae appear to insert directly into the septal wall rather than into a recognizable specialized papillary muscle. The crista supraventricularis presents on the floor of the right ventricular chamber, as seen surgically, between the papillary muscle of the conus below and posteriorly and the pulmonary valve above and anteriorly.

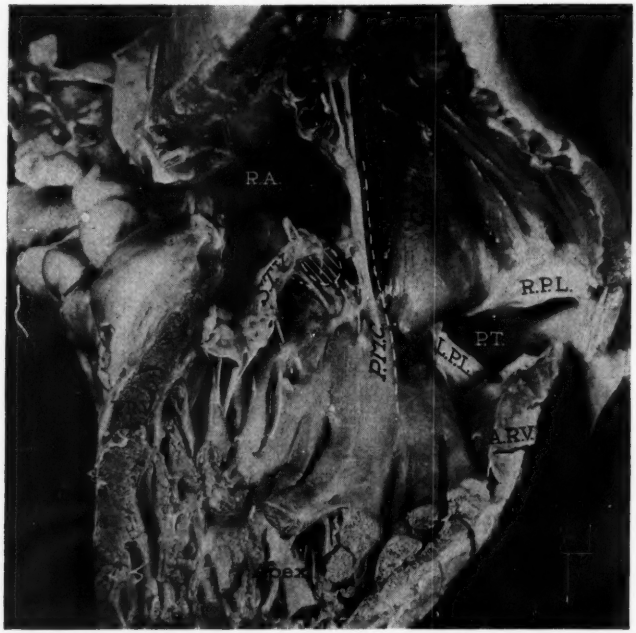

Fig. 1.—Normal heart with right ventricle opened. To view this specimen in the orientation seen at bilateral anterior thoracotomy, the photograph should be rotated 90 degrees counterclockwise. Key: *PT*, pulmonary artery; *LPL* and *RPL*, left and right pulmonary valve cusps; *CS*, crista supraventricularis; *PMC*, papillary muscle of conus ("conus muscle"); *APM*, anterior papillary muscle; *STV*, septal leaflet of tricuspid valve; *RA*, right atrium; *PRV* and *ARV*, posterior and anterior walls of right ventricle. The dotted circle represents the region of the membranous septum.

It is essential to recognize that most of the outflow tract of the right ventricle and the pulmonary valve lie more cephalad and anterior than do the outflow tract of the left ventricle and aortic valve when the heart is in situ. Thus, the crista supraventricularis overlies in part the root of the aorta.

Ventricular septal defects may be classified as indicated in Table I.

TABLE I. TYPES OF VENTRICULAR SEPTAL DEFECTS ENCOUNTERED IN 36 SURGICAL CASES

TYPE OF DEFECT	CASES	
Defect related to ventricular outflow tracts	27	
Inferior to crista supraventricularis	25	
Superior to crista supraventricularis	2	"High"
Defect related to ventricular inflow tracts	11	
Beneath septal leaflet of tricuspid valve	8	
Near apex of muscular septum	3*	"Low"

*In one case the only defects (three) were in this area. In the other 2 cases, defects here were associated with "high" ventricular septal defects.

Defects Related to Ventricular Outflow Tracts and Involving Region Inferior to Crista Supraventricularis.—The region of the right ventricular outflow tract lying inferior to the crista supraventricularis is most commonly involved by ventricular septal defects. Defects in this location, when viewed from the left ventricular aspect, are seen to be immediately beneath the aortic valve cusps (Fig. 2).

These lesions are usually classified as "membranous ventricular septal defects," an inadequate descriptive name since they basically involve a large zone of muscular tissue adjacent to the membranous septum (Fig. 3). The size of the defect itself seems related mainly to the amount of this muscular portion of the ventricular septum that is absent. In some cases, the membranous portion of the ventricular septum is also lacking. In such instances, the septal leaflet of the tricuspid valve and the anterior leaflet of the mitral valve are continuous across the posterior border of the defect.

When the defects in this region are large and involve the septal tissue immediately below the annulus of the aortic valve, the aortic leaflets are seen to overhang the defect (Fig. 4). In all defects with this configuration, the annulus and leaflets of the aortic valve are very near the region where stitches for repair must be placed.

Defects Related to Ventricular Outflow Tracts and Involving Region Superior to Crista Supraventricularis.—The main features of such defects are their close relationship to the pulmonary valve and their lack of extension inferiorly and posteriorly beyond the crista supraventricularis. These defects lie immediately beneath not only the pulmonic valve but also the left aortic cusp (Fig. 5).

Defects Involving Regions of Ventricular Septum Not Related to Ventricular Outflow Tracts.—These defects are located entirely in the muscular wall of the ventricular septum and are related to the inflow or sinus portion of the ventricles. Two categories of such defects are recognized:

1. *Defects lying beneath septal leaflet of tricuspid valve:* These, like the previously discussed types, might be called "high" ventricular septal defects. While they lie in the upper or basal portion of the septum, they are not closely related to the aortic valve when viewed from the left ventricular aspect (Fig. 6). Here they lie near the posterior leaflet of the mitral valve.

2. *Defects in muscular septum near apex:* These are often termed "low" ventricular septal defects (Fig. 7). Fig. 8 shows diagrammatically the location of these types of defects.

Conduction System.—It has been observed at operation that trauma, either by a fixation forceps or a needle point, to the remnant of membranous septum or tissue lying at the postero-inferior margin of the ventricular defect may result in ventricular asystole or heart block for a prolonged period. This suggests that conduction tissue lies in this area.

The detailed relations of the conduction system to ventricular septal defects are yet to be investigated. Nevertheless, observations on conduction disturbances occurring during and subsequent to repair of ventricular septal defects support the view that the conduction system in hearts with such a

Fig. 2.—*a*, Ventricular septal defect related to ventricular outflow tracts and lying posterior to crista supraventricularis, as viewed from the right ventricular aspect. *b*, Same defect from left ventricular aspect. The upper margin of the defect, as viewed from this aspect, is formed by the annulus of the aortic valve.

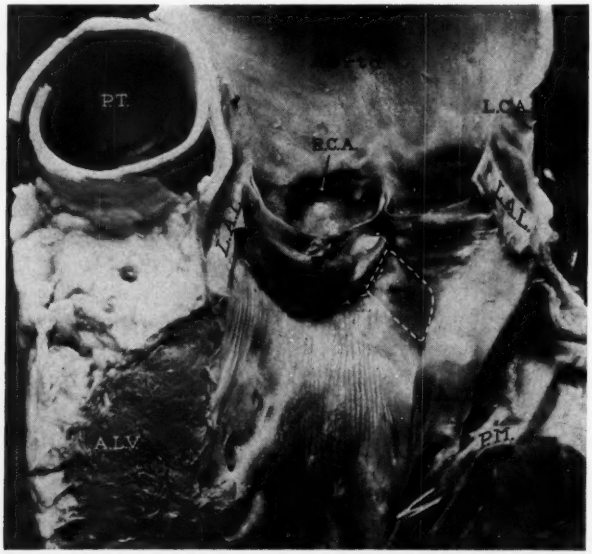

Fig. 3.—Normal heart showing left ventricular aspect of septum. The dotted diamond labeled M is the membranous portion of the septum lying beneath the commissure between the right aortic cusp (*R*) and posterior aortic cusp (*P*). Adjacent is the anterior mitral leaflet (*AM*). The posterior mitral leaflet (*PM*) also is shown.

Fig. 4.—Ventricular septal defect viewed from left ventricular aspect. There is no septal tissue immediately beneath the commissure between the posterior and the right aortic cusps, and these cusps overhang the defect.

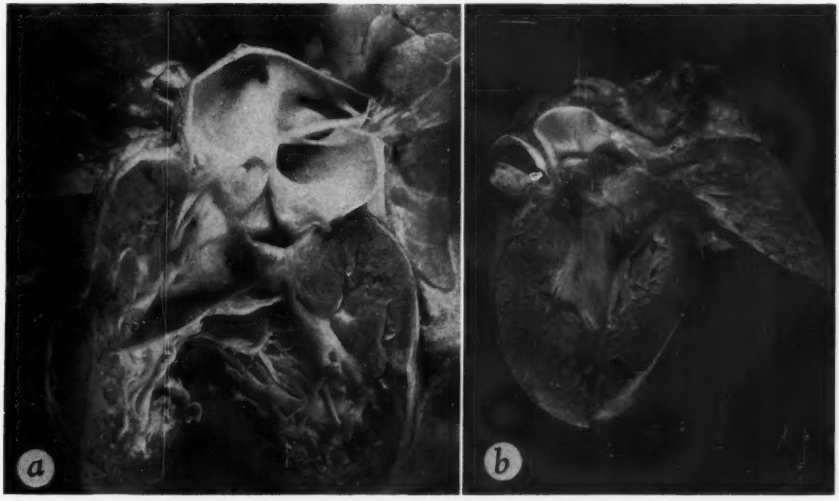

Fig. 5.—Ventricular septal defect anterior to crista supraventricularis viewed from right ventricular (*a*) and left ventricular (*b*) aspect. The defect is immediately beneath the pulmonary valve and just beneath the left aortic cusp.

Fig. 6.—Ventricular septal defect beneath septal leaflet of tricuspid valve. *a*, When viewed from the right ventricular aspect the defect is beneath the septal leaflet of the tricuspid valve. *b*, When viewed from the left ventricular aspect the defect is seen not to be closely related to the aortic valve and is near the posterior mitral leaflet.

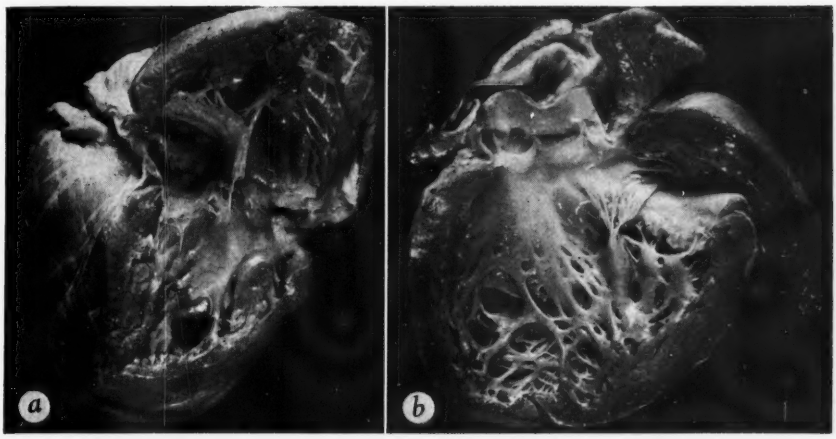

Fig. 7.—Defect near apex of ventricular septum viewed from right (*a*) and left (*b*) ventricular aspects. It is far removed from other important structures.

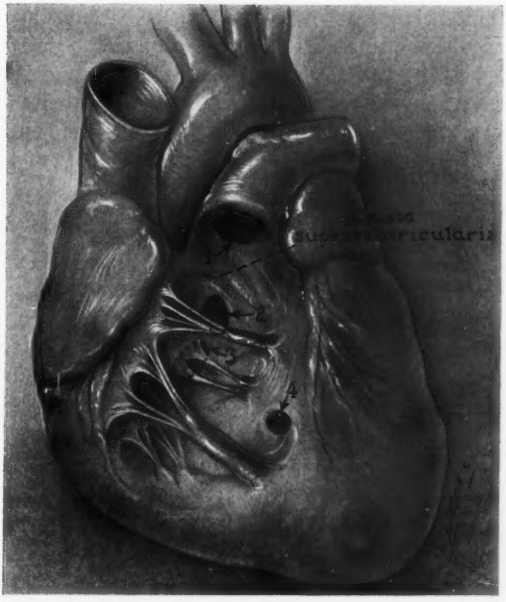

Fig. 8.—Composite schematic drawing of right ventricular aspect of septum indicating the location of defects. Key: *1*, defect immediately beneath the pulmonary valve; *2*, defect related to outflow tracts and inferior to the crista supraventricularis; *3*, defect related to inflow tract beneath septal leaflet of tricuspid valve; *4*, defect near apex of septum.

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defect is placed essentially as it is in normally formed hearts, as described by Widran and Lev.[7] Thus there may be a close relationship of the bundle of His and its right and left branches to those defects that appear in the outflow tract of the right ventricle inferior to the crista supraventricularis. In such defects, the main bundle would be located in the tissue forming the postero-inferior extremity of the defect while the beginning of the right branch of the bundle of His would lie more or less parallel to the inferior edge of the ventricular septal defect, as the right branch runs forward toward the base of the papillary muscle of the conus. Beyond this, it turns downward along the ventricular septum at some distance from the position of the usual type of ventricular septal defect. The region of the base of the papillary muscle of the conus, while grossly related to the position of the right branch, is beyond the location of the origin of fibers of the left branch. The fibers of the left bundle in the normal heart are so placed as to cross toward the left in the tissue inferior to the position of the type of defects under discussion.

The atrioventricular node lying above the base of the septal leaflet of the tricuspid valve and in the lowermost part of the atrial septum at a position anterior to the orifice of the coronary sinus is not as intimately related to the position of ventricular septal defects as are the main bundle of His and its right and left branches.

Defects in the outflow tract of the left ventricle that are superior to the crista supraventricularis and immediately inferior to the pulmonary valve are not closely related to the position of the bundle of His and its branches. Defects in the apex of the septum are not intimately related to the position of the conduction system. Those defects lying beneath the septal leaflet of the tricuspid valve may be related to the position of elements of the right branch of the bundle of His.

MATERIAL

In this presentation, no mention will be made of cases of a ventricular septal defect that is part of the tetralogy of Fallot since the technical considerations are somewhat different in these instances. Cases of common atrioventricular canal are likewise excluded.

Thirty-six patients who underwent surgical treatment of a ventricular septal defect between March 1, 1955, and April 15, 1956, comprise the basis for this report. The most commonly encountered type of defect was that related to the ventricular outflow tracts, inferior to the crista supraventricularis (see Table I). Nineteen of the twenty-five defects of this type seemed to be completely above the chordae tendineae to the papillary muscle of the conus while six were both above and below these chordae. In 12 of the 25 cases, the aortic leaflets could be seen prominently in the operative field as they overhung the defect. This indicates very little septal tissue, if any, remaining between the annulus of the aortic valve and the defect. In 10 cases, there was no posterior rim of residual membranous septum and the posterior border of the defect was formed by a membrane representing the continuous portion of the septal leaflet of the tricuspid valve and the anterior mitral leaflet.

There were associated defects in some cases (Table II). Associated atrial septal defect, patent ductus arteriosus, and mitral stenosis were corrected at the time the ventricular septal defect was repaired.

TABLE II. ASSOCIATED ANATOMIC DEFECTS IN 36 SURGICAL CASES OF VENTRICULAR SEPTAL DEFECT

ASSOCIATED DEFECT	CASES
Atrial septal defect	4
Persistent left superior vena cava	3
Patent ductus arteriosus	1
Congenital mitral stenosis	1
Right aortic arch	1

COMPLETENESS OF REPAIR

Since many of the patients were small children, it has been difficult to urge early postoperative cardiac catheterization upon them. Even postoperative catheterization is not an infallible method for identifying small residual shunts unless done with extreme care and accuracy. In evidence of this is the fact that adult patients who have not been operated on and who have very loud systolic murmurs characteristic of maladie de Roger may have a left-to-right shunt so small as to be detectable only with difficulty by the usual catheterization techniques.

All would agree that complete absence of the systolic murmur of ventricular septal defect is excellent evidence of complete repair. The presence of a short, rough, Grade 1 or 2 systolic murmur over the precordium after operation does not prove the presence of a residual shunt, but the presence of such a murmur does not allow the patient to be considered as having a proved complete repair until the result is confirmed by cardiac catheterization. A patient with such a murmur but with a normal indicator-dilution curve as determined in the operating room after repair, a quiet heart and clinical improvement as manifested by rapid growth, gain in weight and disappearance of symptoms presumably has at least a satisfactory repair and perhaps a complete repair. A persisting loud systolic murmur, combined with an indicator-dilution curve determined immediately after repair and indicating persisting left-to-right shunt even though small, gives clear evidence of a residual shunt. When death occurs postoperatively, necropsy allows clear decision as to completeness of repair.

TABLE III. RESULTS OF REPAIR OF VENTRICULAR SEPTAL DEFECT IN THE ENTIRE SERIES OF 36 CASES IN WHICH VARIOUS TECHNIQUES WERE USED AND IN THE LAST 25 CASES, IN WHICH THE PRESENTLY EMPLOYED TECHNIQUE WAS USED

RESULT	VARIOUS TECHNIQUES (ALL 36 CASES)	PRESENT TECHNIQUE (LAST 25 CASES)
Complete repair	20	18
Satisfactory repair (may well be complete but not yet so proved)	7	4
Known residual shunt	9	3

The completeness of repair for the present series as adjudged by these criteria is indicated in Table III. Techniques used include closure by direct suture, by a now abandoned technique using compressed polyvinyl-formal

(Ivalon) sponge, and by the presently employed method. The last technique has been developed because of disappointment with occasional incomplete repair by the methods used earlier. That this technique, to be described, has been a distinct advance is indicated by the data portrayed in Table III; with it there have been only three instances of known residual shunts. One was in a patient with multiple ventricular septal defects, a very small one of which was closed imperfectly by a single stitch which has allowed a small residual shunt.

This experience emphasizes the importance of meticulous technique in the actual repair of these defects, made difficult in some instances by proximity of valvular structures and by problems of exposure of certain areas of the lesion. These obstacles must be overcome if the goal of complete, secure closure for each patient is to be reached.

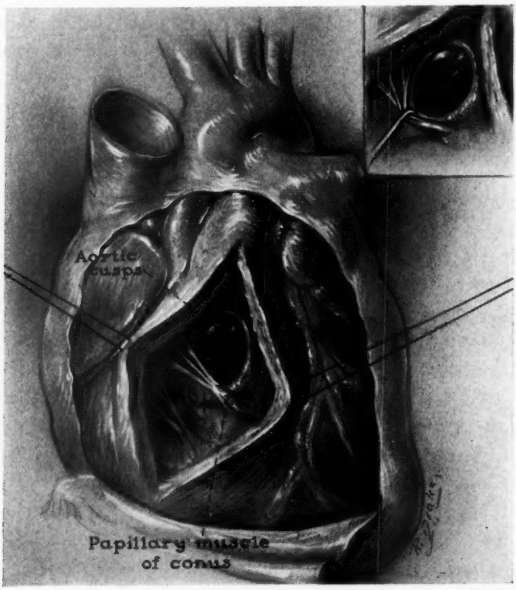

Fig. 9.—Drawing of ventricular septal defect inferior to crista supraventricularis as seen at operation.

TECHNIQUE OF REPAIR

Since most defects seen surgically are related to the ventricular outflow tracts and are postero-inferior to the crista supraventricularis, closure of this type of defect will be described. Such defects are more difficult of secure repair than are other types. The technique described has been used in the last 25 cases of this series.

After the function of pumping and oxygenation of blood is taken over by the pump-oxygenator unit, the right ventricle is opened widely. The ventricular septal defect is identified and its relation to the aortic and tricuspid

leaflets noted (Fig. 9). The postero-inferior area is scrutinized to determine whether this is composed of a remnant of membranous septum or whether the remnant is absent and instead the leaflets of the mitral and tricuspid valves are continuous across this area. The proposed placement of stitches is considered so as to avoid placing them directly posterior in a position which seems to invite conduction disturbances. Should ventricular asystole or heart block occur as a stitch is being placed, warning has been served that this stitch should be removed and not tied down.

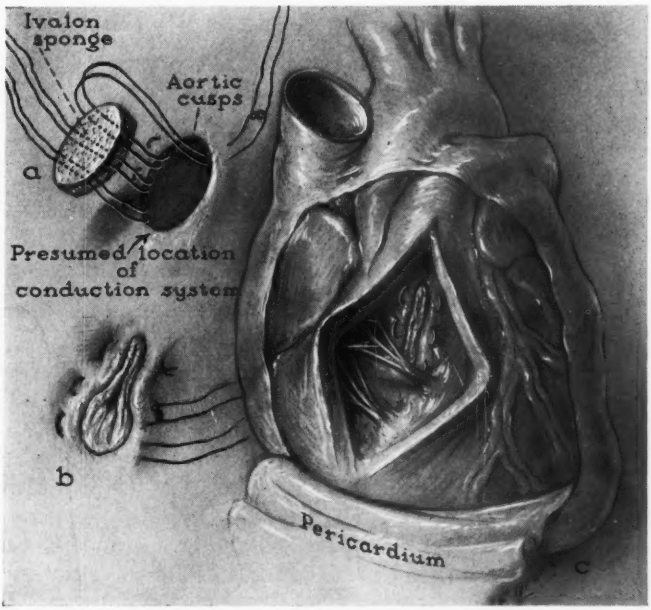

Fig. 10.—*a*, *b*, and *c*, Repair of ventricular septal defect using noncompressed polyvinyl-formal sponge. Note in *a* the proximity of aortic cusps to the stitches. Misplaced deep stitches in this area may tear an aortic cusp. The area probably occupied by the conduction system should be avoided in stitching.

On occasion, chordae tendineae attaching to the papillary muscle of the conus partly overlie these defects. In such instances, they are retracted. Mattress sutures of 2-0 silk are then placed for repair so that they grasp one side of the defect, pass through an appropriately trimmed piece of noncompressed polyvinyl-formal (Ivalon) sponge and then through the other edge of the defect (Fig. 10). As the mattress stitches are tied down, they bring the edges of the defect together against the interposed sponge to obtain a secure closure. The line of closure is parallel to the direction of outflow from the right ventricle but is at right angles to the direction of outflow from the left ventricle. The opportunity for distortion of aortic valve leaflets is minimized because of this.

Sometimes the placement of these sutures in the inferior aspect of the right edge of the defect, as viewed from the right ventricular side, is difficult. This area may be formed by the tricuspid valve ring itself and is partially overhung by the septal leaflet of this valve. The most inferior pair of mattress sutures may, in such instances, be inserted into this margin of the defect through the tricuspid ring, penetrating it from the right atrial side. These sutures then go through the sponge and left edge of the defect to be tied down. If the defect lies completely beneath the septal leaflet of the tricuspid valve, it is often best to place all the mattress sutures into the right margin of the defect by suturing through the tricuspid valve ring (which forms this margin of such defects).

After the mattress sutures have been tied down, the repair is carefully scrutinized for residual tiny defects. If present, these must be securely closed by additional stitches. Likewise, after the obvious large defect is closed, the septum should be examined for smaller defects elsewhere. Bronchial blood coming to the left side of the heart will tend to appear through these small additional defects after the large one has been blocked. Such defects are closed by direct suture.

COMMENT

The use of noncompressed polyvinyl-formal sponge in the manner described has been exceedingly satisfactory. The technique must be slightly altered for other types of "high" ventricular septal defects, although the basic principles are the same.

Direct suture of the "high" defects was employed in the first 3 of the 36 cases and in one later case which seemed ideally suited to it. Perhaps direct suture could be used more often than it was employed in this series but experience with the third case in which it was used discouraged its frequent application. In this case, the patient died eight hours after operation and necropsy disclosed that the suture line had partially disrupted, making the repair very incomplete. This, plus other experiences in the operating room, has lent conviction to the belief that the living ventricular septum allows tight stitches to cut through rather readily. In these large lesions, the use of a securely anchored polyvinyl-formed sponge to help fill the tissue defect allows a snug repair without the stitches having to be drawn too tightly.

SUMMARY

A thorough understanding of the surgical anatomy of ventricular septal defect is essential to the successful treatment of it. Particular attention must be given to the variation in location of the defects. Exact knowledge of the relation of the defect to aortic leaflets, mitral and tricuspid leaflets, and the bundle of His is of inestimable value. The technique that has proved most satisfactory to date is described. Emphasis is placed on the necessity for precision in the repair to avoid damaging adjacent structures and to ensure absolute and permanent closure.

ADDENDUM

Further experience since submission of this manuscript has caused the authors to adopt the routine use of potassium-induced cardiac asystole in the repair of ventricular septal defects. It is to be pointed out that the details of the actual closure with this technique are somewhat different than those described in this paper. This is noted in an attempt to avoid misleading those interested in this field as to the method of repair now being employed by us.

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Discussion

DR. C. W. LILLEHEI, Minneapolis, Minn.—I rise to heartily endorse the very fine presentation of Dr. Kirklin, and to comment upon one of the ancillary problems related to closing septal defects that was not touched upon by either Dr. Kirklin or Dr. Warden due to time limitations. I refer to the occurrence and persistence of complete heart block in occasional patients following intracardiac surgery. I think it would be presumptuous to say that this problem has been solved, but we have learned a good deal about its management by the empiricism of trial and error and, since many more surgeons will be undertaking these procedures in the near future, it may be of value to summarize briefly some of the information of value that has been obtained by this painfully slow method of inquiry.

The incidence of complete heart block has been about 12 per cent in the patients having ventricular septal defects repaired.

There are probably two important elements inherent in the causation of this complication; one is the placement of the stitches so as to injure or interrupt the main conduction pathways. I'll say a further word about that in a moment. The other element is a little more difficult to define precisely, but is related to the status of the patient's cardiac reserve. That is, among the patients having ventricular septal defects closed, there have been 11 cases of complete heart block and all have occurred in the patients with severe pulmonary hypertension; that is, in the group where the pulmonary pressures exceeded 70 to 80 per cent of the systemic pressures. Thus, there is an increased predisposition to block where the myocardial reserve is less. In fact, in an occasional instance in such a patient, we have seen complete heart block occur before any stitches had been placed in the heart. The same consideration applies to the patients with tetralogy of Fallot; in those in whom the myocardium is severely hypoxic or the cardiac reserve is lessened greatly, heart block is more likely to occur.

The conduction tissue has a high glycogen content and the pathways may be clearly demonstrated in the fresh heart by painting the septum with Lugol's solution.

(slide) These color photographs, prepared by Dr. Rivkin of our staff, show the anatomy of the conduction pathways in a fresh canine heart stained in this fashion. The atrioventricular node is shown here at the base of the interatrial septum lightly craniad to the coronary sinus. The atrioventricular bundle is seen as it proceeds horizontally forward, reaching the inferior edge of the membranous portion of the interventricular septum where it divides into left and right branches (bundle of His). Note in this photograph how quickly the left bundle, as it descends toward the apex, fans out into a diffuse broad sheet of fibers lying subendocardially. This diffuseness is a favorable anatomic circumstance for the surgeon since it makes it less likely that a single stitch will encompass the entire left bundle.

In this photograph of the right side of the septum in the same heart, the right bundle branch is seen as a continuation of the atrioventricular bundle. It passes undivided, subendocardially, to the anterior papillary muscle at which point it commences to branch out over the right ventricle.

Now, in the typically located ventricular defect due to absence of the membranous septum, this inferior margin of the membranous defect (apex of the muscular portion of the septum near the anterior margin of the tricuspid septal leaflet) is the most vulnerable area since a single stitch deeply placed here may encompass both right and left bundles causing complete heart block. In this area, we prefer placing our closure stitches on the right ventricular aspect of the defect margin, parallel with the defect margins, and not too deeply (so as not to penetrate to the left side). Thus, we often get a right bundle branch block, but this is innocuous since the rate and rhythm of the heart remain unchanged. If complete block is seen to occur with placement of a particular stitch, we generally remove the offending stitch and replace it in a slightly different place. This maneuver may or may not be of value but if it is not we continue with the closure stitches regardless, since our primary objective, if the patient is to survive, must be to close the defect.

The treatment that has proved lifesaving in those patients, in whom complete block persists, has been constant vigilance together with the use of the drug Isuprel (isopropyl levoarterenol). Death in patients with complete block occurs because the rate falls so low that the systemic pressure and coronary circulation deteriorate due to the diminished output, and cardiac arrest occurs. Successful treatment requires keeping the heart rate above this critical level (about 80 to 100 per minute in surgical patients) for a sufficient time interval until sinus rhythm recurs or the circulatory system has had a chance to readjust to the slower rate. In our first 3 patients we used epinephrine, ephedrine, atropine, and lactate for this purpose. They all produce some temporary beneficial effects, but all 3 patients eventually died due to their block.

We were a little slow to try Isuprel because an intravenous preparation was not available commercially. However, it is available now from Winthrop Stearns Company. We use a 1:50,000 solution and give 0.5 to 1.5 c.c. intravenously to infants, and up to 2 and 3 c.c. to adults per dose. With this dose the heart rate will accelerate and the effect often lasts for fifteen or twenty minutes. In some cases, there may actually be a sinus rhythm which may persist for a variable period, but often relapses again into complete block. In other cases, Isuprel merely keeps the heart rate at an acceptable level, somewhere above 90 to 100 beats per minute. Three or four hours after surgery, when the patient's condition has stabilized somewhat and the heart block has still persisted, we usually switch to the use of Isuprel linguets, 5 to 15 mg., given rectally. This form of administration is more satisfactory for long-term therapy inasmuch as an effect is produced within about thirty minutes which lasts from two to four hours. In all cases these patients must be monitored constantly by electrocardiography. If sinus rhythm recurs, as it has in all of our survivors, Isuprel should be continued in gradually diminishing quantities for another ten to fourteen days to prevent sudden deaths due to recurrence of com-

plete block. Occasionally, Isuprel has appeared to lose its effectiveness, and this has been restored by combining the Isuprel with relatively small doses of intravenous molar lactate. Utilizing Isuprel, 5 of the last 8 patients developing complete heart block after closure of their ventricular defect have been saved, and all have recovered a sinus rhythm before leaving the hospital.

DR. ALFRED BLALOCK, Baltimore, Md.—Dr. Edward Churchill said at a recent meeting, when called upon to talk, that Senior Members should be seen but not heard. I cannot resist the temptation to congratulate the people who have presented these papers. It is fascinating work, and I am sure that when you become as old as I am you will look back on this era of the beginning of intracardiac surgery with a great deal of pleasure. Certainly this meeting has been a most interesting one and I have listened very intently. In leaving for home I do not know quite which attack to prefer. I am inclined to think that, whereas magnificant results have been obtained by the group in Minneapolis, probably the Kirklin modification of the Gibbon apparatus is today the best procedure to follow. I would predict, however, that in ten years this would seem as outmoded as some of you regard some of the procedures done ten years ago.

I was interested to hear John Kirklin at the beginning of his paper call these "corrective procedures." I truly believe this to be a better term than that used in Minneapolis, "curative procedures." My particular interest is in the tetralogy of Fallot, some of which may be partially corrected by previous techniques. In speaking of that lesion alone at the moment, when there is a mortality, as I understand Dr. Varco reported in New York recently, of 45 per cent, and when the patients have been followed for very short periods, I do not think—and I will admit to being old-fashioned—that this should as yet be called a curative procedure. I do not wish to indicate that I am not fascinated and thrilled by these results which have been reported not only this afternoon but throughout the meeting. I might say, however, that developments are going so fast that the faster I run, the further I get behind.

DR. JOHN W. KIRKLIN (Closing).—I am sure that everyone in this audience is indebted to Dr. Blalock for his comments on this problem. Certainly those of us who are young and enthusiastic do not at times have the privilege of looking at things in their proper perspective.

I would like to second what Dr. Lillehei has said about the conduction system. In my anxiety and nervousness I forgot to point out something on the slide.

(slide) Dr. Lillehei has already mentioned this, but just to emphasize the point which we both feel is very important: in this inferior angle is the presumed location of the conduction system. Certainly we feel, although the evidence is perhaps not conclusive, that one should not put a stitch in that area. I think quoting results as evidence is always open to considerable question, but for what it is worth, in the last 25 cases closed by this repair, we have had no heart block. In the cases done prior to that, 10 or 11, we had three heart blocks. One patient died on the sixth postoperative day. The other 2 survived with the Isuprel technique mentioned and resumed a normal sinus rhythm. Certainly it is a complication that we would all like to avoid. Thinking of those methods employing purposeful cardiac arrest, I suppose one question that must be asked when one is employing this technique is whether the advantages outweigh the disadvantage of not knowing when to withdraw a stitch should block occur.

Dr. Gerbode asked me to say a word about common atrioventricular canal. That of course is a totally different type of defect with regard to technical considerations. It is an operable defect. We have had the opportunity to operate on 9 of these patients and 6 of them have survived operation; we have lost 3. We cannot really say for certain but, for what it is worth, it seems to me that this is the place where I would almost be willing to say it is absolutely necessary to use a prosthesis. There is a large tissue defect and it would seem that repair by direct suture must always be under considerable tension, and one wonders if that does not predispose in some way to heart block.

THE USE OF THE HEART-LUNG APPARATUS IN HUMAN CARDIAC SURGERY

F. D. Dodrill, M.D., Norman Marshall, M.D. (by invitation), Jan Nyboer, M.D. (by invitation), C. H. Hughes, B.S. (Med. Biol.) (by invitation), A. J. Derbyshire, Ph.D. (by invitation), and A. B. Stearns, M.D. (by invitation)
Detroit, Mich.

DIFFERENT types of heart-lung machines are now in clinical use.[1, 2] All these types of mechanical devices have as their function the removal of venous blood as it returns to the heart, its arterialization and return to the aortic system, thereby taking over the function of the heart and lungs. There are, at the present time, two points of view as to the method of by-passing the heart and lungs using the varius types of machines. First of these involves the circulation and arterialization of a portion of the total blood volume. This is referred to as the low flow principle in extracorporeal circulation.[3] The desirable amount of blood to circulate in the low flow principle has not been definitely decided upon and has varied from 20 c.c. per kilogram of body weight per minute up to 50 to 60 c.c. per kilogram of body weight per minute. The normal blood volume in man is approximately 78 to 84 c.c. per kilogram of body weight, being slightly higher for males than for females.[4] Patients with left-to-right intracardiac shunts have greater than normal blood volumes because of the great amount contained in the dilated pulmonary vessels. The second principle in the extracorporeal circulation is the circulation of the total blood volume. There are advantages and disadvantages to both plans in the extracorporeal circulation. This manuscript deals with the circulation of the total blood volume, its complete arterialization, and the maintenance of normal systolic and diastolic arterial pressures.

LOW FLOW PRINCIPLE

One of the chief reasons why the low flow principle is being carried out in extracorporeal circulation is the fact that it has not been possible, heretofore, to handle the total blood volume, to completely arterialize it, and to maintain normal pressures. While this has been discouraging over the years, it seems more hopeful that this plan will become a practical procedure. The advantages of a low flow circulation are that this can be handled more easily, that the apparatus needs to be less formidable and, more importantly, that the injury to the blood may be less because the amount which is circulating outside the body at a given time is less. There seems to be no definite advantage in the low flow principle aside from these facts.

From the Research Division of Harper Hospital and the Research Laboratories Division, General Motors Corporation.

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There are obvious disadvantages to the low flow principle. The circulating blood volume is less and, therefore, the oxygen which is transported to the tissues is definitely less. This is the chief and most important disadvantage of the principle. With low flows, the arterial pressures are lower than if normal flows are used. While these disadvantages may not be so great as to lead to death, it would seem better if the subject could be maintained on normal physiologic standards. In young patients, circulation, of course, can be shut off for short periods of time. If one then circulates a small amount of blood which is properly arterialized, this period of occlusion can be extended for varying periods of time. It seems, therefore, that the use of the low flow principle, *as the goal,* in extracorporeal circulation is not satisfactory.

While many successful operations on patients have been performed with open cardiac surgery maintaining substandard physiologic processes, it would seem logical to believe that, if normal physiologic values were maintained, the over-all results would be superior.

TWO INNOVATIONS

For a description of the apparatus used, the reader is referred to our previous publications.[5, 6] However, two innovations have been made in the plan of the operative technique. The first of these consists in a so-called exchange transfusion in which the patient's blood is partially removed together with some donor blood, placed in a properly prepared flask beside the operating table while the patient is maintained on the remainder of his own blood together with donor blood. At the conclusion of the pumping time, the used blood is washed out to a certain extent, while the original blood is returned to his system, following which the pump arterializer is disconnected from the system. The second innovation is in the return of the blood to the aortic system. This is accomplished by suturing onto the side of the descending aorta, just below the origin of the left subclavian artery, a homologous aortic graft of sufficient size and caliber to permit the return of the total circulation to the aorta without its having to go through a narrow orifice such as the left subclavian artery.

Exchange Transfusion.—

One of the major drawbacks to extracorporeal circulation has been the injury to the blood. Some of these injuries are known while some are unknown. It has been customary to think of the injury to the blood only in terms of hemolysis of the red cells. Inasmuch as the injury to the blood may be due not only to the actual physical trauma but also to complex changes in the chemical and enzyme systems, the amount of hemolysis in the blood is one of the ancillary manifestations of the total over-all alteration in the blood constituents. One of the discouraging complications, therefore, in extracorporeal circulation has been the development of the bleeding diathesis following the operative procedure. This bleeding diathesis may be prevented in different ways. It may be possible to add certain substances to the blood which will prevent its alteration during the extracorporeal circulation. Less traumatic machines may be constructed which will more nearly simulate the

heart and vascular system. Finally, the injured and deleterious components of the blood may be removed, or partially removed, in a process of exchange transfusion similar to that which is performed in erythroblastosis fetalis. In this condition, the infant's blood is incompatible with its own system. The noxious elements may be partially removed by process of exchange transfusion whereby a quantity of the patient's own blood is removed and replaced with compatible donor blood. It is not necessary or possible to remove all the patient's blood in this condition but, rather, the noxious elements are diluted down to the point where they are sublethal.

When one operates on a small patient who has a blood volume of 500 to 1,000 c.c. of blood, there is almost complete removal of his blood in the extracorporeal circuit. At least, one removes a large part of the patient's blood and replaces it with mixed donor blood. While this can be done effectively in most cases, it is probable that certain complications in cross matching and incompatible blood might possibly enter into the picture. On the other hand, if a fairly good portion of his own blood is preserved and returned to his circuit at the end of the operation, these possible complications might be obviated.

At the conclusion of any pumping period, it is probable that the serum potassium is elevated in all cases. This degree of elevation is not necessarily parallel, or dependent upon, the amount of hemolysis in the blood. This can be greatly reduced by the exchange transfusion at the conclusion of the procedure.

While it may not be necessary for short runs, such as thirty to forty minutes, one never knows exactly how long will be required to repair the defect. Moreover, the time is now at hand when more complex defects may be repairable. The fact of the matter is that there are no nontraumatic heart-lung machines available at the present time.

When we are ready to place the patient on the pump arterializer, he is given heparin solution in the amount of 4 mg. per kilogram of body weight. At the conclusion of the procedure and after the cannulae are removed from the patient, he is given protamine sulfate in the proportion of 1.2 to 1 of the amount of heparin. The clotting mechanism has returned to normal in all cases within forty-five minutes from the time the protamine has been administered. In these patients who have been carefully studied as to their clotting times postoperatively, we have seldom observed the so-called rebound phenomenon, that is, having the clotting times repeatedly return to normal but in a few hours rise again to a prolonged clotting time. The actual explanation for this rebound phenomenon has not been forthcoming but we suggest that a possible explanation of this is the postoperative breakup of the red cells which, in itself, may release additional heparin.

The advantages of this exchange transfusion are so great and so protean that time and space do not permit a complete statement of its advantages. Suffice it to say, that the advantages are great and that the possibilities for good have not been completely utilized. Moreover, aside from the mechanical heart-lung work, this may be of some use in other forms of clinical surgery in which uncontrolled bleeding from unknown causes ensue.

It has occurred to us, therefore, that since complex alterations may be taking place in the blood during extracorporeal circulation which may not be fully understood at the present time, to remove the noxious elements by performing a so-called exchange transfusion. With the pump arterializer connected to the patient, the technical aspect of the procedure is extremely simple.

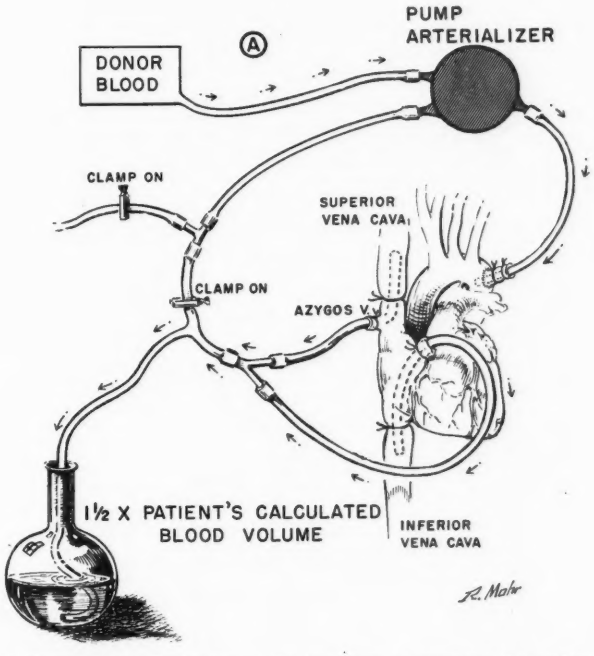

Fig. 1.—A diagrammatic illustration of the technique of removing the patient's blood.

Technique of exchange transfusion: With the pump arterializer connected to the patient as for extracorporeal circulation, the apparatus is turned on and blood is allowed to enter the aorta under normal pressure while the venae cavae blood is allowed to collect in a properly prepared vessel by the side of the operating table (Fig. 1). Normal aortic arterial pressures must be maintained in order that the volume of blood put into the aorta may be equal to that which is removed from the venae cavae. It is amazing that under these circumstances the volume of blood which is collected by the side of the operating table practically always equals that which is put into the arterial system. Obviously, there is mixing of the donor and patient's blood during the procedure. It has been our practice to remove one and one-half times the calculated volume of the patient's blood. This is an arbitrary figure but has been found to be satisfactory in the amount of the patient's actual blood removed. Using radioactive serum albumin for blood volume determinations, it has been found that the removal of one and one-half times the patient's volume results in the removal of from 35 to 40 per cent of his own actual blood

(Fig. 2). At the conclusion of the extracorporeal circulation, this volume of blood is passed through the pump arterializer one time and returned to the patient's circulation following which the pump is disconnected from the vascular system (Fig. 3).

EXCHANGE TRANSFUSION

case	Calculated Bl. volume	Amount removed	Amount of Pt's. own Bl. removed
1	2000cc	3000cc	1140cc
2	3000	4000	1400
3	1200	1800	720

Fig. 2.—Blood volume studies on 3 patients. Radioactive sotope technique using iodinated human serum albumin.

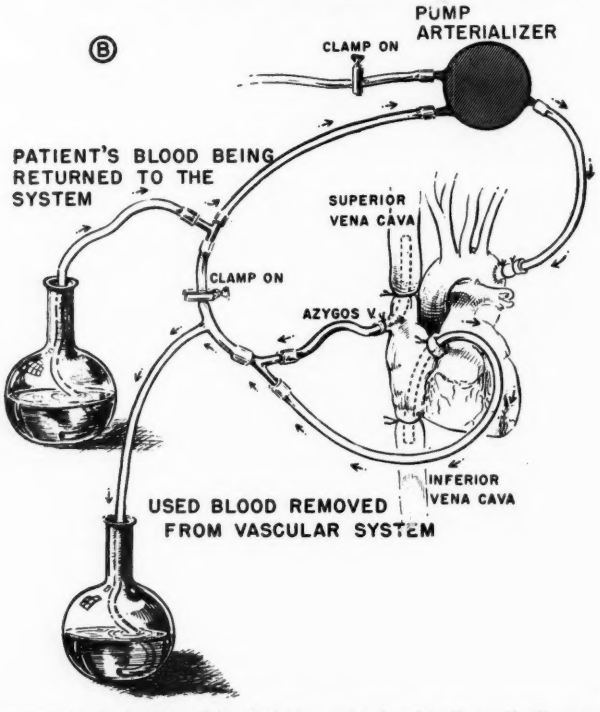

Fig. 3.—Diagrammatic illustration of the technique of returning the patient's own blood to his vascular system and the simultaneous removal of the used blood.

Return of the Blood to the Aorta.—

It is our belief, based upon several years' experience with extracorporeal circulation, that it is not possible to maintain good arterial pressures by returning the subject's blood volume by way of the subclavian artery. This is

obviously true when one considers that the orifice of the subclavian artery may be only from one third to one fourth the actual size of the aortic valve through which the blood volume has been ejected in the subject. Also, the return of the blood to the aortic system by way of the small subclavian orifice is obviously one of the major points in the turbulence of the blood. Moreover, the return of the blood through a small orifice dissipates the pulse pressure to a large degree. While it has not been definitely shown that a pulse pressure is necessary, we believe that it is desirable to maintain it, if possible. Likewise, the maintenance of normal arterial pressure both systolic and diastolic during the extracorporeal circulation has not been proved to be absolutely necessary to life for short periods. While many patients throughout the country have been successfully operated upon with good recovery with a low arterial pressure during the extracorporeal circulation, the desirable thing

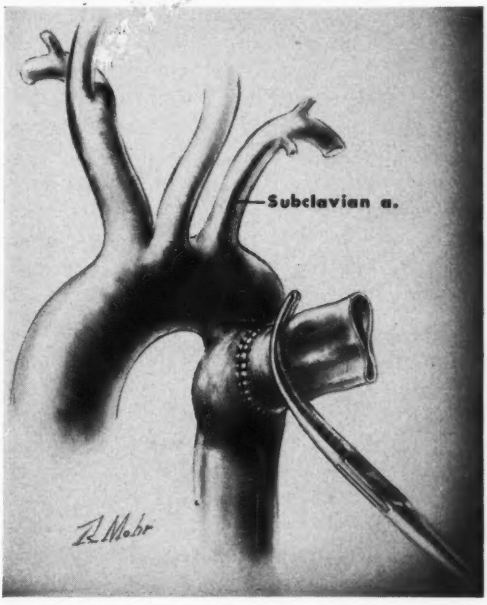

Fig. 4.—Diagrammatic illustration of an aortic graft sutured onto the side of the descending aorta.

to do is to maintain normal systolic and diastolic pressures according to our normal standard physiologic values. This can be accomplished only by the return of the blood to the aorta through a larger orifice than the left subclavian artery, in fact, the orifice should be essentially and nearly equal to the aortic valve of the particular subject being operated upon.

We are, therefore, making a linear incision on the lateral surface of the aorta just below the origin of the left subclavian artery, suturing onto this an arterial graft of proper size and caliber (Fig. 4). This orifice is usually made as large as the approximate diameter of the aorta itself. We recognize the

fact that this adds something to the operative procedure but we are willing to accept this disadvantage for the obvious advantages one obtains in having complete control over the extracorporeal circulation once it is connected. This is our first attempt to return the blood through a large orifice. There may be more simple and practical ways of doing it than the suturing on of a graft on the side of the aorta.

APPLICATION TO PATIENTS

We have applied this new technique to a group of 6 patients. The types of anatomic defects operated upon have all been interventricular septal defects with the exception of one patient with tetralogy of Fallot, who incidentally survived. They have been maintained on normal physiologic standards insofar as we have been able to determine. These physiologic checks include the electrocardiogram, electroencephalogram, intra-arterial and central venous pressures together with the usual blood checking data such as arterial oxygen saturation, plasma carbon dioxide, and pH. The pumping period has varied from twenty-one minutes to fifty-two minutes' time. The average has been twenty-five to thirty minutes. The prolonged pumping period in the one patient resulted because of the fact that, after the ventricular septal defect in the high membranous septum had been closed by method of suture, there was a rupture in a separate portion of the septum aside from the suture line and a large defect resulted. This required an additional thirty-five minutes to repair. In some of the instances, the physiologic data have been more normal while the patient was on the pump arterializer than immediately before he was put on it.

In these six attempts, using this method, 3 patients have died postoperatively giving us an operative mortality of 50 per cent. Two of these patients were operated upon earlier when the method of inflating the lungs with helium was not currently practiced and both of these patients succumbed to pulmonary atelectasis (Fig. 5). A third patient died from intrapericardial hemorrhage forty-eight hours, postoperatively. All of these patients have temporarily recovered from the procedure and have lived at least forty-eight hours.

VOLUME FLOWS

Using this plan of returning the blood to the aorta through a large orifice, we are able to maintain perfectly normal blood flows. It is our experience that in patients with congenital heart disease with a left-to-right shunt that the total circulating blood volume is in excess of normal values. In all of the cases to date, it has been found that the circulating volume has been from 90 to 100 c.c. per kilogram of body weight per minute using radioactive albumin. This has actually been the blood flow in all cases which has been determined by a Wier-type flowmeter.

One of the amazing phenomena or observations following this plan is that the amount of postoperative drainage is extremely small. Of the 6 patients, all of whom survived at least forty-eight hours postoperatively, in only

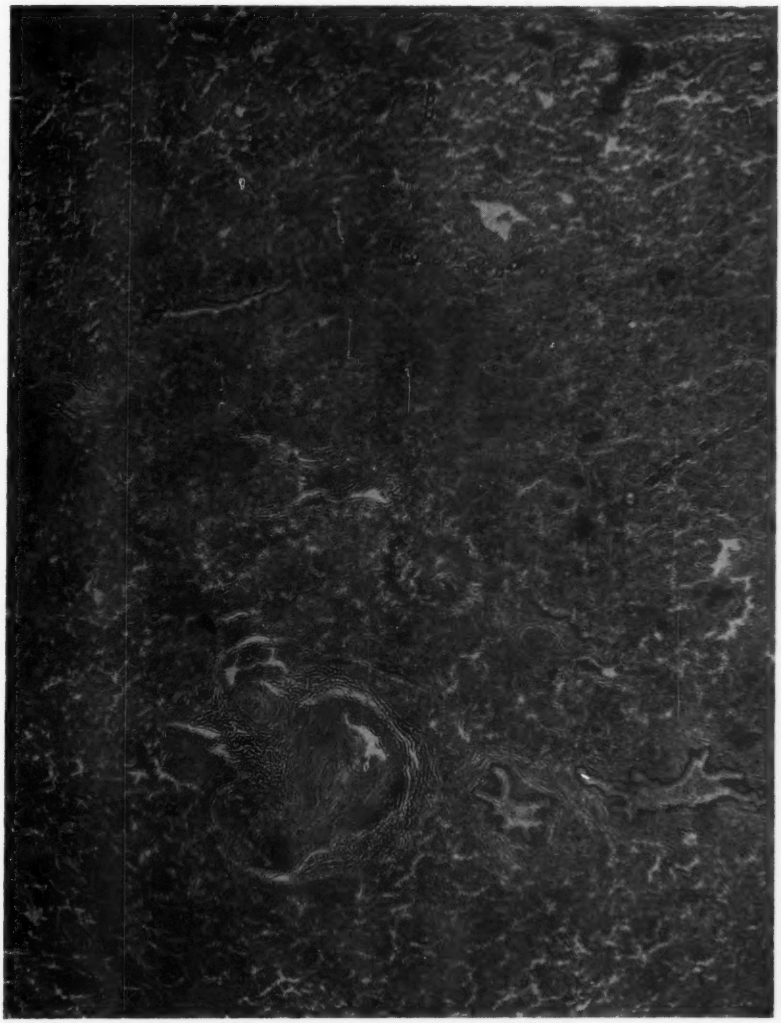

Fig. 5.—Microscopic section of postoperative atelectasis of the lung.

one was there a drainage of from 300 to 350 c.c. of serum. In all the others the drainage was so minimal that it was hardly measurable. This, in itself, indicates to us that the enzyme mechanisms of the blood are essentially normal.

TYPE OF SUTURE USED IN REPAIR OF INTERVENTRICULAR SEPTAL DEFECT

Fig. 6 illustrates a figure-of-eight type of suture which was used to approximate the septal defect. While this may not be accepted by all as superior to a single interrupted suture, it is the impression that a figure-of-eight stitch properly placed actually has less pressure upon a given point of the tissue than if a single interrupted one were used.

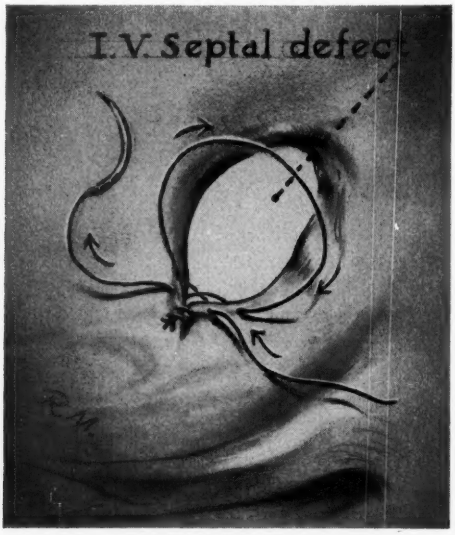

Fig. 6.—Illustration of a figure-of-eight type of suture for repair of interventricular septal defects.

PHYSIOLOGIC DATA USING THE EXCHANGE TRANSFUSION AND THE LARGE ORIFICE IN THE AORTA FOR THE RETURN OF THE CIRCULATION

Fig. 7 illustrates the patient's blood presssure before, during the exchange transfusion, and after being completely on the pump arterializer. It is seen at a glance that the systolic and diastolic pressures both during the exchange transfusion and subsequent to it, during the pumping period, are entirely normal, being about 120/80 mm. Hg. It has been the experience of most investigators that during the pumping period the arterial pressure tends to drop somewhat during the period. It has been our experience, on the other hand, that, uniformly, the pressure rises as the pumping period continues and stabilizes near the preoperative level. This is illustrated in Fig. 8. One unphysiologic phenomenon which we have observed during this procedure is the rise in the venous pressure. This rise in venous pressure does not necessarily mean that too much blood is being pumped. It may be adequately explained

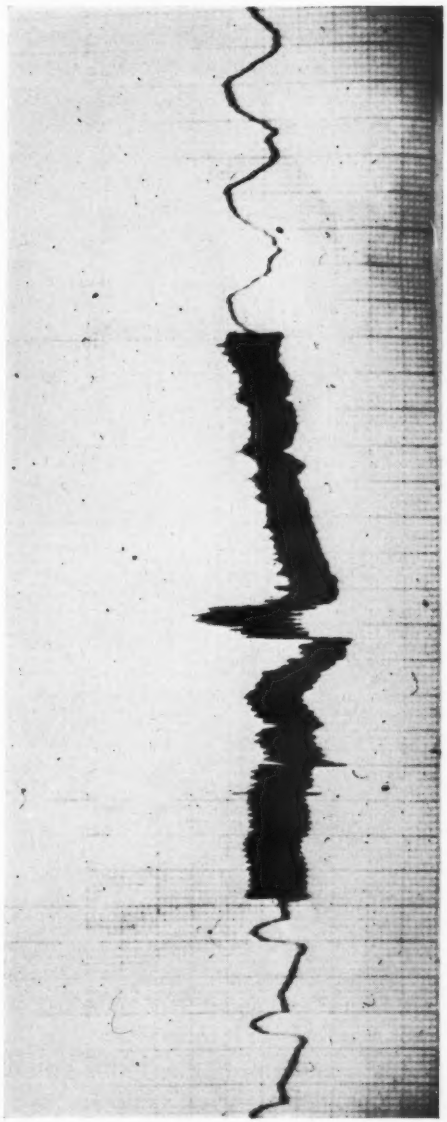

Fig. 7.—The actual record of the arterial blood pressure before pumping, during the exchange transfusion, and after being completely on the pump arterializer.

by the fact that the catheters in the superior and inferior venae cavae are much smaller than the lumina of these vessels and must necessarily be so since, from the time that the catheters are put into the venae cavae, there must be some flow around them to maintain circulation until the pump can be connected. The blood, therefore, is withdrawn through a narrow orifice as it enters the venae cavae. We would, therefore, accept considerable rise in the venous pressure such as 30 to 40 mm. Hg. If, however, the pressure should rise too high, we would be inclined to remove some blood from the extracorporeal circulation, set it aside on the operating table and return it to the system at the conclusion of the procedure. This has actually been done on only one occasion.

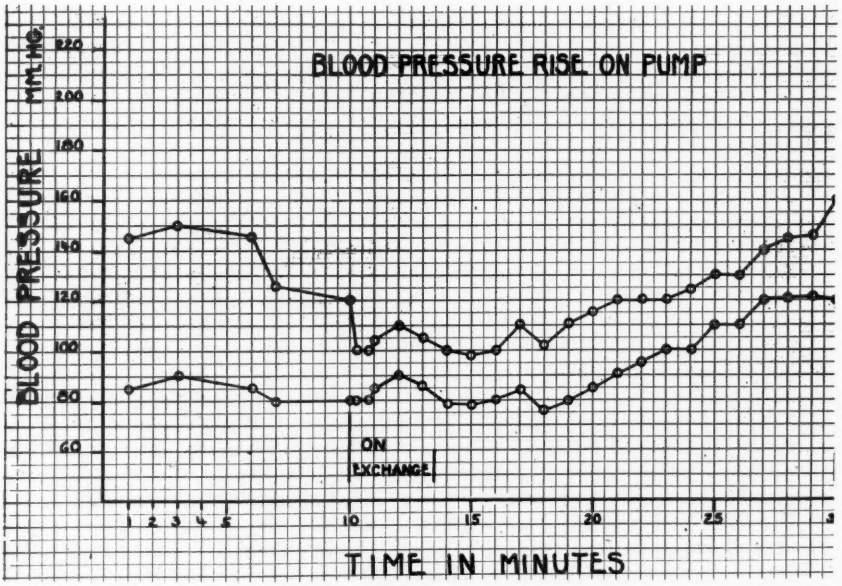

Fig. 8.—Illustrates the rise of the arterial blood pressure during the pumping period until stabilized at the preoperative level.

Arterial Saturations.—Fig. 9 shows the saturation of both arterial and venous blood during the extracorporeal circulation. During all cases it is readily seen that the saturation is 100 per cent or nearly so. Likewise, the arterial-venous difference in saturation is satisfactory.

Plasma Carbon Dioxide.—The table in Fig. 10 illustrates the pCO_2 during the extracorporeal circulation. These normal values for the pCO_2 can be maintained with the bubble type of arterializer only, if some carbon dioxide is used for the perfusing gas. We have shown in a previous publication[5] the desirability of adding carbon dioxide to the perfusing gas. At present, we are using 96 per cent oxygen and 4 per cent carbon dioxide.

The pH Values.—During our previous investigative work over the years, it was our experience that during the pumping period the pH of the blood tended to drop below normal values. This has been thought of and has been spoken of as due to metabolic acidosis and was thought to be due to insufficient circulation. It is interesting to note that, with our plan of total extracorporeal

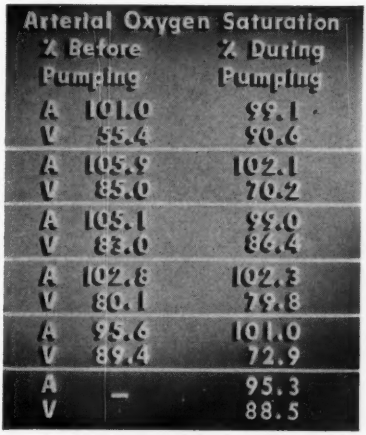

Fig. 9.

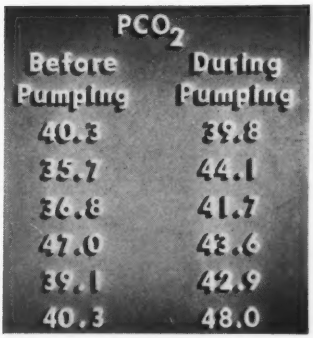

Fig. 10.

Fig. 9.—Oxygen saturation determinations by the Van Slyke method. Oxygen saturation of arterial and venous blood before pumping and toward the end of the pumping period in 6 patients.

Fig. 10.—PCO_2 determinations by the Van Slyke method. PCO_2 before pumping and toward the end of the pumping period in 6 patients.

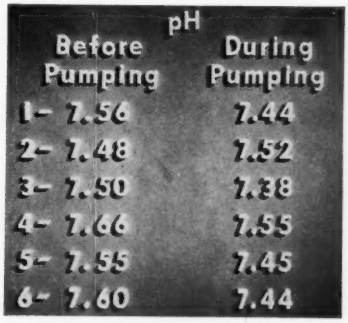

Fig. 11.

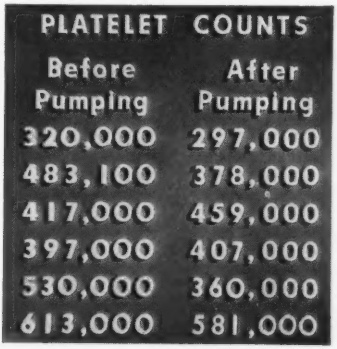

Fig. 12.

Fig. 11.—The pH determinations by the Beckman pH meter. Before pumping and toward the end of the pumping period in 6 patients.

Fig. 12.—Platelet counts before and after extracorporeal circulation in 6 patients.

circulation maintaining normal systolic and diastolic blood pressures, this fall in pH does not take place. Fig. 11 illustrates the pH taken during the pumping period and illustrates a perfectly normal value. To our experience, this can only be obtained by the maintenance of normal systolic and diastolic pressure and a maintenance of normal flow.

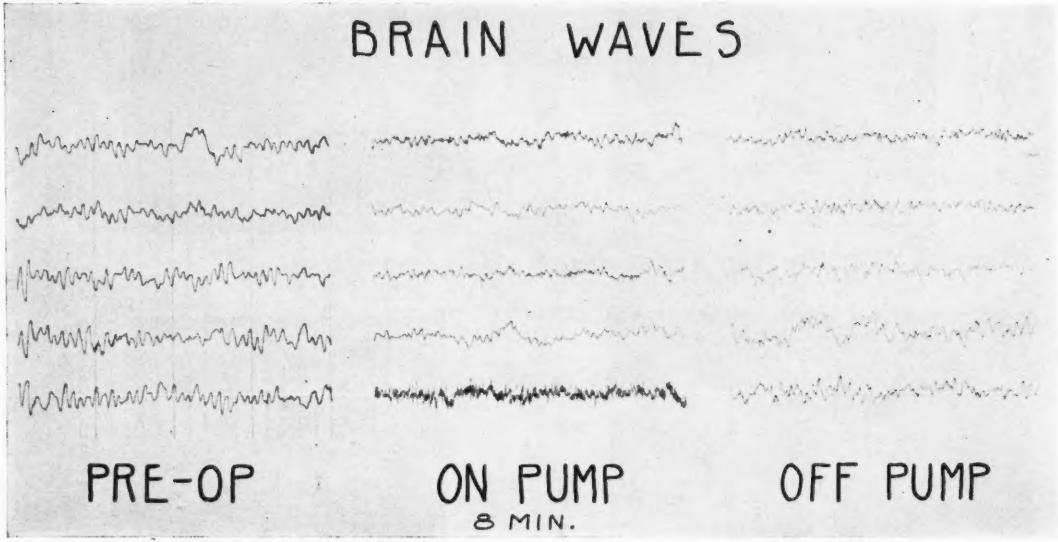

Fig. 13.—The electroencephalogram before, during pumping, and immediately after pumping.

Blood Platelets.—The blood platelets were counted before and immediately after the pumping time. No destruction is seen (Fig. 12).

Electroencephalogram.—The electroencephalogram was taken on all cases. This was found to be normal except for a brief period, lasting two to three minutes during the exchange transfusion, when there was some abnormality (Fig. 13).

SUMMARY

Two innovations in extracorporeal circulation are presented. The first involves the so-called exchange transfusion in which a portion of the blood is removed and placed on the operating table while the patient is maintained on donor blood. At the conclusion of the procedure, the original blood removed is returned to his system following which the pumping device is disconnected. Indications are that the blood is essentially normal following this procedure.

The second innovation involves the suturing of an aortic graft on the side of the aorta producing a large orifice through which the arterialized blood is returned to the system.

Using this plan of procedure, the total blood volume of any subject can be properly arterialized and the systolic and diastolic arterial pressure maintained according to normal standards. Indications from various physiologic data show that the total body physiology is likely to be more normal using this plan of circulation.

By this procedure, 6 patients have been operated upon employing this technique. During the procedure, various data have been recorded which include the arterial pressure, central venous pressure, the effects on the electroencephalogram and the electrocardiogram. Various blood chemistry data have been presented. All data obtained indicate a more normal body physiology can be obtained than has heretofore been illustrated.

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DISCUSSION

DR. JOHN H. GIBBON, JR., Philadelphia, Pa.—In the first place, I would like to say that, as Dr. Dodrill well knows, I am in complete agreement with his point of view that we should attempt to have as nearly normal conditions as possible during the period of open intracardiac surgery, just as during any operative procedure. In other words, we would like to avoid hemolysis of the blood, we would like to have the blood adequately saturated with oxygen, we would like to have a normal blood flow, and we would like to avoid either acidosis or alkalosis. I could not be more heartily in favor with that point of view. Cer-

tainly his figures of venous oxygen saturation would indicate that he had an adequate blood flow during the period of by-pass. I would be the first to agree that probably more things happen to the blood passing through an artificial circuit outside the body than the obvious things which can be detected by measurement, such as the breaking down of the cellular elements of the blood. Many of us have been studying the blood and have found great difficulty in putting our fingers on anything specific, except the fact that the leukocytes and platelets tend to decline in concentration. The effect upon the erythrocytes can be detected by studying the amount of free hemoglobin in the plasma.

I was sorry Dr. Dodrill did not indicate the level of free hemoglobin in the plasma of the donor blood. I presume it was reasonably high or he would not have employed an exchange transfusion. I take it also that the amounts of free hemoglobin in the plasma which he reported are the figures after replacement with the recipient's own blood, and not those of the donor blood which was used during the by-pass. Many of us have reported the levels of free hemoglobin in the plasma, the decline in white blood cell count, and the decline in platelet count. The decline in platelet count is not severe enough to be responsible for postoperative hemorrhage in our experience, and the decline in white cell count returns, in the course of several hours, to a normal level.

Might I comment on some of yesterday's papers? I was extraordinarily interested in, and pleased to see, all the interest shown in the Forum program yesterday, and I regret that I left before the conclusion of the program as I assumed there would be no discussion. I merely wish to make one or two comments. I think that Melrose's suggestion of arresting the heart by injecting a potassium salt into the aorta between an aortic clamp and the aortic valves, should be an extraordinarily helpful one. The demonstration that all that is necesssary is to remove the clamp and wash out the coronary vessels in order to restore a normal beat, to my mind is very exciting and should prove very useful in intracardiac surgery of any sort, but particularly with by-pass through an extracorporeal circuit. Cardiac arrest should decrease the amount of cardiac venous blood which returns to the cardiac chambers, which will be helpful from a technical standpoint.

I was very much interested in Dr. Clowes's presentation. Kolff, ten years ago in Holland, told me that he was sure the artificial kidney would serve just as well as an artificial lung. I told him I rather doubted it. It has turned out that it is an extraordinarily difficult problem, as Dr. Clowes showed yesterday, and he is to be congratulated for his persistence in working with it. It is hard enough to get sufficient gas exchange between the fluid blood and the atmosphere with a free blood-gas interface, and if you interpose anything between the blood and the gas it is going to slow down the process considerably. Nevertheless, as Dr. Blalock said, ten years from now we are all going to look back upon our present devices as though they were old T-model Fords. The future of the artificial lung, which is the important part of the extracorporeal circuit (the pumping is easy), may well be what George Clowes has shown. If one can avoid a free blood-gas interface and still accomplish an adequate gas exchange, then an enormous step in advance will have been taken and we will have got rid of a lot of problems. I hope very much that he and others will stay with this problem and finally come up with something that resembles the pulmonary capillaries, difficult as that may be.

DR. F. D. DODRILL (Closing).—I wish to thank Dr. Gibbon for his discussion. The plasma hemoglobin on these patients with the total heart-lung by-pass, using the large volume flows, has varied from 100 to 150 mg. per cent for thirty minutes' duration. With the exchange transfusion at the conclusion of the procedure, we remove about 50 per cent of the increased plasma hemoglobin. In addition, we are removing approximately the same amount of deleterious substances, whatever they may be.

It is obvious that the blood is one of the most complex substances with which the surgeon deals. Twenty years ago a textbook of physiology might devote about two pages to the blood-clotting mechanism where as now as many as fifty pages might be devoted to this subject. Inasmuch as this whole field of blood physiology is rapidly expanding, this simple matter of diluting or removing a portion of these deleterious elements will, I hope, be of help to others during similar types of work.

THE TREATMENT OF MITRAL INSUFFICIENCY BY THE PURSE-STRING TECHNIQUE

Initial Clinical Application

Robert P. Glover, M.D., and Julio C. Davila, M.D. (by invitation)
Philadelphia, Pa.

THE very considerable success in the surgical treatment of stenotic valvular disease of the heart in recent years has emphasized the urgency for the development of equally satisfactory methods for the correction of valvular insufficiencies. The mitral valve offers untold opportunity for the study and correction of its incompetent state just as this same valve did eight years ago when stenosis was first successfully relieved by surgical means.

Although a systolic murmur at the apex of the heart in the mitral valve area has long been recognized as perhaps the commonest of all valvular findings, many have considered for years, and have so stated, that mitral insufficiency is a relatively benign lesion and as such is rarely the cause of untoward clinical symptoms leading to death. The authors initially were prepared to accept this viewpoint during the early years of our surgical experience with mitral commissurotomy. Shortly, however, it became apparent all too frequently that the coexistence of mitral stenosis and insufficiency presented a clinical picture and a surgical problem which was quite different from that posed by the pure, tight, stenotic valve. This impression was strengthened when a review of our first consecutive 500 mitral commissurotomy cases revealed that the commonest incidence of either operative or late postoperative death occurred in those patients who either had pre-existing associated insufficiency unrelieved or worsened by commissurotomy or those in whom a pure state of stenosis was surgically transformed into a case of significant mitral insufficiency. It became even more obvious when, as time passed, a number of mitral valves were explored under the impression that stenosis was the predominant lesion only to find that very little if any stenosis was present and that indeed the entire syndrome of deterioration had been secondary to a relatively pure state of mitral insufficiency.

At the outset, it was recognized that insufficiency of the mitral valve was undoubtedly a more insidious lesion than was stenosis but that in time it created even more serious effects to the myocardium of the left heart (left

From the Department of Thoracic and Cardiac Surgery at Presbyterian and Episcopal Hospitals, and The Cardiovascular Research Laboratory at the Presbyterian Hospital, Philadelphia, Pa.

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ventricle and atrium) than did its stenotic counterpart. Reports in the literature, together with some of our own more painful clinical experiences, indicated that the acute production of mitral insufficiency of significant degree was attended by almost immediate disaster both to the experimental animal and to the human patient. As an adequate experimental and/or clinical study had never satisfactorily been made and reported, concerning the pathophysiologic changes in chronic mitral insufficiency, this aspect of the over-all problem remained obscure except by inference and impression. Therefore, in 1952, on the thoracic surgical services at Episcopal and Presbyterian Hospitals and in the Cardiovascular Research Laboratory at Presbyterian Hospital, a concentrated program for the study of mitral insufficiency was inaugurated and has been carried out continuously to the present time.

PATHOPHYSIOLOGIC CONSIDERATIONS

It goes without saying that before any concentrated study of the pathophysiologic derangements of an organ or, as in this case, a heart valve can be undertaken, a complete and thorough understanding of the normal anatomy and physiology must be basic knowledge. The first year of this program therefore was spent studying the normal anatomy of the mitral valve and its leaflets, the mitral annulus, the myocardial configuration at the atrioventricular juncture and the relationships of all other structures adjacent to the mitral valve and its annulus.[1] As many of the hearts examined were the seat of mitral valve disease, both stenosis and insufficiency, certain concepts as to the pathology of mitral insufficiency were gained together with a knowledge of its intricate anatomy.

With but minor variation it was learned that three basic features were present in the regurgitant mitral valve. *First,* and foremost, rheumatic valvular disease in such cases results in an absolute loss of valve tissue. As the aortic (septal) and the mural leaflets of the mitral valve contract within their substance they lose contact with each other and are no longer able to coapt during ventricular systole. As a rule this contraction is greatest in the mural leaflet which frequently becomes almost nonexistent. *Second,* and concomitantly, the chordae tendineae attached to the valve cusps become contracted, shortened, hypertrophied, and agglutinated tending further to fix the valve leaflets in a state of partial immobility. This effect increases the inability of the valve leaflets to coapt but it must be emphasized that even though contracted the leaflets (particularly the aortic) usually retained considerable pliability. Thus, during ventricular systole the mitral orifice remains open at a time when closure is necessary to properly and effectively direct the left ventricular outflow into the aorta. These two pathologic findings can obviously be termed *"an absolute loss of valvular occluding elements."* A *third* pathologic entity is almost routinely present. In a sense it might be considered a result of the first two although actually it too may appear concomitantly with the first two pathologic findings. As a result of the fact that the mitral valve leaflets cannot adequately close with each ventricular systole, a considerable vascular stream is propelled into the left atrium. For a considerable time it

would seem, the left atrial chamber and its myocardium absorbs the effect of this jetlike shock by gradual dilatation and hypertrophy in so far as its myocardial elasticity will allow. The systolic jet and backpressure being intermittent and not sustained throughout the cardiac cycle (as it is in stenosis), the left atrium itself absorbs the total effect for an indefinite period and thus protects the pulmonary vascular bed and right heart from an increased pressure. In consequence, however, the left atrium enlarges constantly eventually to assume tremendous aneurysmal saclike proportions. Simultaneously the left ventricle, laboring to maintain an effective systemic output under impaired hydraulic conditions, is overworked. It responds by gradual dilatation and hypertrophy. Thus, as ventricle and atrium enlarge, the common point of their continuity—the mitral annulus or atrioventricular ring—does likewise, thus dilating the circumferential base to which the valve leaflets are attached

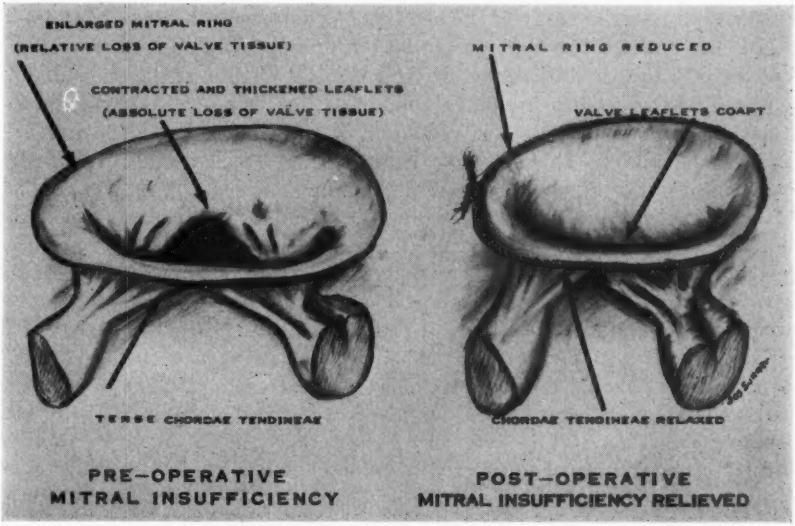


Fig. 1.—This diagram indicates the three major factors of pathologic deformity which contribute to mitral insufficiency. The drawing on the left shows the valve and ring prior to circumferential constriction. The drawing on the right indicates the effect of the purse-string in accomplishing reduction of annular size, relaxation of chordae tendineae and valve leaflets and making possible more complete coaptation of leaflet edges.

and hinge. Thus, the *third* factor—dilatation of the mitral ring—is added to the production of mitral insufficiency. Now, not only are the contracted valve leaflets unable to close the mitral orifice but, with annular dilatation, the orifice to be closed becomes larger and a vicious cycle has been set up which cannot terminate other than by complete myocardial exhaustion. The concept, therefore, is not merely that the left heart myocardium wears out from mitral insufficiency of stationary amount and degree but rather is faced with an ever increasing load due to slow, gradual but constant enlargement of the mitral ring and orifice. Two factors are therefore at play—myocardial overwork and an ever increasing load (Fig. 1).

With this concept in mind it seemed obvious that any surgical correction must be directed toward the relief of all three of these factors which play a part in the production of mitral valve insufficiency. Heretofore, and for that matter even to the present time, most of the methods advocated for the correction of mitral insufficiency took into account only the fact that there was loss of valve tissue. Attempts were, therefore, made to repair or replace or add to the missing valvular occluding elements. Techniques employing prosthetic devices,[2-8] grafts of pericardium or veins,[9-16] or methods of suturing the leaflets to each other were tried by several surgeons.[2, 9, 17] These procedures have been applied clinically to large numbers of unsuspecting patients despite the fact that little or no preliminary experimental work or evaluation was carried out. Further, in published reports, there is no objective evidence that these presumed corrective measures were effective. The clinical results have suggested, however, that such methods were unsatisfactory and disappointing and the eventual result in many of the patients has not been reported.

To the authors there seemed to be but one approach. As little could be done directly to the valve leaflets or their chordae tendineae, the one remaining approach was to modify the mitral annulus.[1, 18-20] A possible solution seemed to be based on the principle of reduction of the size of the orifice which the valve must occlude. This obviously could only be accomplished by controlled constriction of the atrioventricular ring or annulus (see Fig. 1). The concept was doubly attractive because it implied the avoidance of intracardiac foreign bodies and utilized to the full extent all available functional valvular occluding elements which remained, however scarred or puckered they might be. Further, if one accepts the observation that mitral insufficiency progressively increases as the annulus continues to dilate secondary to enlargement of the left heart chambers, circumferential annular constriction with its fixation of the mitral ring should abruptly terminate the increasing degree of regurgitation even though it may not be possible to totally abolish all of the original insufficiency. This factor is of vast importance, for no operation, however cleverly conceived, can be expected to completely remove in a routine manner all trace of insufficiency in the markedly diseased valves which one encounters in such cases. Any operation, therefore, which merely attacks a localized segment of the mitral annulus cannot be expected to overcome indefinitely the problem of future annular dilatation.

Two years were then spent in the animal research laboratory exploring the methods whereby an effective and at the same time innocuous circumferential suture could be placed about the mitral annulus. As might be expected, innumerable problems arose. The experiences encountered and the technical solutions employed for the final development of the operation now called "mitral purse-string" have been reported in previous communications. These data have clearly shown that experimental mitral insufficiency produced by almost every conceivable method can be routinely corrected by total annular constriction and documentary objective proof of this contention has been recorded. In addition to its ability to overcome the majority, if not all, of the mitral insufficiency present, it has been shown that mitral purse-string

does not produce mitral stenosis, does not injure the effective myocardium, does not injure the coronary arterial system, and when properly placed does not cut through into the lumen of the left atrium or produce appreciable conduction defects. Animals, in whom mitral insufficiency has been produced experimentally and corrected by total controlled circumferential constriction of the annulus, have presently been followed for well over two years and some 20 such animals are being followed up to five years to determine the eventual status of a suture so placed.

TECHNIQUE OF MITRAL PURSE-STRING

The technique in its final form, presently being employed in clinical mitral insufficiency, is outlined as follows: The patient is placed in the right lateral decubitus position with the left side up. The usual left posterolateral incision is made, entering the pleura through the bed of the resected sixth rib. Ribs five and seven are divided posteriorly if additional exposure is needed. This is often the case when one is dealing with the massively enlarged heart. The inferior pulmonary ligament is divided to the level of the inferior pulmonary vein ensuring adequate exposure posterior to the left ventricle. The pericardium is divided vertically, well posterior and parallel to the phrenic nerve, to expose the entire left heart from the level of the aortic arch to the diaphragm. In the far-advanced case (and even in more minor cases in which there is considerable mitral insufficiency), the left atrium will be tremendous in size, often aneurysmal, extending inferiorly to the diaphragm and well over to and even beyond the right border of the cardiac silhouette. A massive systolic thrill can be felt everywhere over the greatly dilated left atrial myocardium and this thrill can be traced out over the pulmonary veins into the pulmonary parenchyma. Indeed, on occasions the enlarged and hypertrophied left ventricle seems almost insignificant in size as it protrudes from this tremendous atrial mass. The heart may give the appearance of a large acorn with the left ventricle being the body of the nut and the left atrium being the caplike stem. In relatively pure mitral insufficiency, no other cardiac thrills will be felt. In associated mitral stenosis of significant degree, a short diastolic thrill will be felt over the left ventricle near its apex. Routinely, the ascending aorta and the root of the aorta at the aortic valve ring should be palpated and compressed for evidences of aortic valve disease, for ofttimes patients, with a marked degree of mitral insufficiency, will present clinically some evidence of a systolic murmur at the base often leading to the misdiagnosis of associated aortic stenosis. This is perhaps occasioned because the massive left atrium is in contact with the ascending aorta and transmits its systolic impulse or, as may also happen, the regurgitant stream through the open mitral valve is directed toward the interatrial septum and the jet impulse is transmitted upward to the aorta in this manner. Intracardiac pressure recordings are then taken in the left ventricle, left atrium, pulmonary artery, and aorta. It is extremely important that such pressure tracings be obtained for only in this way can one be objectively sure during surgery that cardiac hemodynamics have been affected by the operative procedure.

When the presence of significant mitral insufficiency has been ascertained in this manner the purse-string suture to be used is then prepared. A rectangular piece of pericardium is resected and sutured like a sleeve over the midportion of a long, double strand of standard, narrow cotton or umbilical tape. This sleeved portion need be no longer than 2 inches for it covers only that portion of the circumferential suture which lies in the transverse sinus from the point of emergency of the suture from the right atrium to the level of the circumflex coronary artery. Our experimental work suggests that it is important to bolster this area for should there be any tendency for the suture to cut through the annulus it will always be in this area. The use of the sleeve as described has prevented any such occurrence both experimentally and clinically.

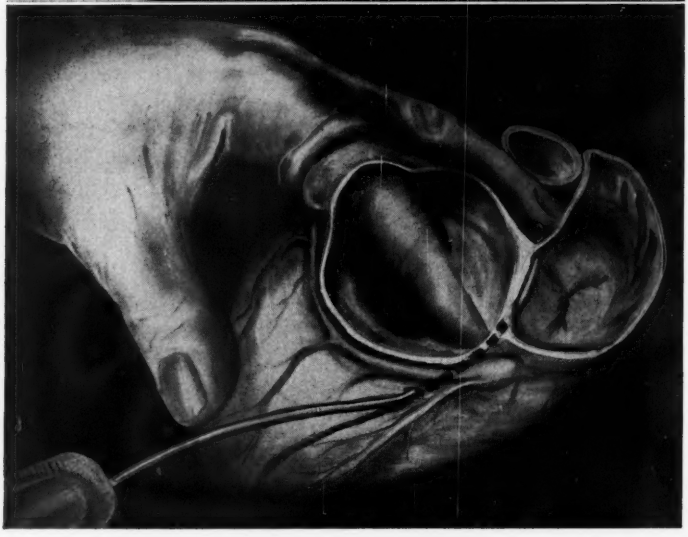
As the suture is eventually applied immediately to the ventricular side of the great cardiac vein and circumflex coronary artery, the circumflex coronary artery just distal to the origin of its septal branch is partially isolated by tunnelling under the vessel so that the suture can eventually be drawn through (Fig. 2). The left atrial appendage is then purse-stringed in the usual manner, clamped and its tip opened. The right index finger is introduced into the left atrium and thorough exploration carried out. The usual finding in predominant mitral insufficiency, despite considerable leaflet contraction, scarring and thickening, is an aortic or septal leaflet of reasonable size and pliability unable to contact its opposing mural leaflet both by virtue of its small size and the fact that it is held in a partially open state by contracted and foreshortened chordae tendineae. The posterior or mural leaflet, as a rule, is more badly diseased, considerably more contracted, and frequently presents merely as a thickened ridge emerging from the myocardium itself. Often there will be very minor agglutination of the two leaflets at the extreme angles but unless one or the other of the commissures is very well developed in the typical stenotic fashion, commissurotomy is not necessary or advised. The orifice of the mitral valve under these conditions will usually admit two to three fingers side by side and the mitral annulus can be recognized as an outer ring four to five fingerbreadths in diameter. Regurgitation of blood with each ventricular systole can be easily appreciated not as a swift, narrow jet, as is found in predominant stenosis, but more as a broad tongue or sheet of swiftly moving fluid. When insufficiency of this magnitude is present, it is impossible to estimate the amount of regurgitation present merely by finger palpation and appreciation. In predominant mitral insufficiency with little stenosis, the valve leaflets will usually have a considerable degree of pliability and very little if any calcium. With more major degrees of concomitant stenosis, one then finds more thickening, immobility, and calcific deposition. This is in line with the theory long held and often expressed by the author that calcium deposition is greatest in those valves which are immobile and are subjected to the incessant trauma of vascular flow pressure impinging upon leaflets partially fixed at right angles to the flowing stream of blood such as is seen in the valve deformity of stenosis.

Having ascertained the conditions present, the actual passage of the purse-string is then effected. The right index finger is removed and replaced by the left index finger. This finger is extended diagonally across the lumen of the left atrium and its tip comes to rest just on the atrial side of the crotch formed by the posteromedial angle of the valve orifice (Fig. 3). At this point the most medial aspect of the annulus can be appreciated. An assistant then gently elevates and rotates the left ventricle to a very minor degree *slightly* to the right. The diaphragm and the posterior leaflet of the pericardium are depressed. The operator can then clearly see the posterior aspect of the left atrium and left ventricle and can readily visualize the course of the posterior descending coronary artery and vein and the angle which these vessels form with the great cardiac vein and coronary sinus. The tip of the Davila* needle is then applied to the myocardium just below and to the left of this angle and advanced beneath the vessels and overlying fat which is invariably present. The intracardiac finger can immediately feel the pressure application of this needle and the further advancement of the needle is guided entirely by the intracardiac finger. The needle is passed in a tangential manner to the finger tip and myocardium and directed posteromedially and superiorly into the right atrium (see Fig. 3). This distance is as a rule no greater than 1 to 1.5 cm. or possibly 2 cm. in the largest of left atria. The needle or ligature carrier is entirely outside of the left atrial myocardium but pierces the wall of the right atrium as described. The assistant now allows the heart to return to its normal position, this period of very minor dislocation being never more than but a few seconds. The operator's left middle finger is then inserted into the depths of the transverse sinus where it comes to rest in the angle formed by the divergent walls of the two atria at the point where they combine to form the interatrial septum. The tip of the needle lying in the right atrium is directed to hug the interatrial septum and is guided onto the tip of the middle finger. At this point it lies between the intracardiac index finger tip and the tip of the middle finger. It is then gently thrust into the depths of the transverse sinus through the wall of the right atrium right at its juncture with the wall of the left atrium and interatrial septum. The needle is stabilized in this position and moved no further so that no kinking, displacement, or compression of the heart occurs. The wire which runs through the entire length of the Davila needle is then advanced and, as it does so, the eye of the needle is carried up from the depths of the transverse sinus out into the operative field where it can easily be threaded (Fig. 4). This feature of the needle prevents displacement or trauma to the heart during the act of threading. The end of the previously prepared purse-string is loosely tied to the eye of the needle, the wire withdrawn back into the depths of the transverse sinus and fixed again to the curved needle. The entire needle is withdrawn in parabolic reverse carrying with it the purse-string. The purse-string suture is pulled out from the posterior aspect of the heart until the sleeved portion of the purse-string suture has been snugged against the depths of the transverse sinus, very effectively tamponading the opening made in the right atrium by

*Available at The George P. Pilling and Son Company, Philadelphia, Pa.

Fig. 2.

Fig. 3.



Figs. 2 to 7.—The technical details of the procedure are illustrated. As can be seen from these diagrams, the greatest part of this operation is actually done under direct vision from the outside of the heart. The exception to this is the maneuver of placing the medial or septal portion of the "purse-string." This is the crucial step of the technique and its execution must be thoroughly mastered in dogs before any clinical attempts should be made. Once this step of the operation is learned the procedure is quite simple.

Fig. 2.—Tunneling under the proximal portion of the circumflex coronary artery. This is done at a point about one cm. beyond the take-off of the anterior descending coronary artery.

Fig. 3.—Placement of the septal portion of the suture. The intra-atrial left index finger, and the left middle finger placed in the transverse sinus guide the needle. It is essential that the operator be clearly oriented in a three-dimensional sense as to the pertinent anatomy.

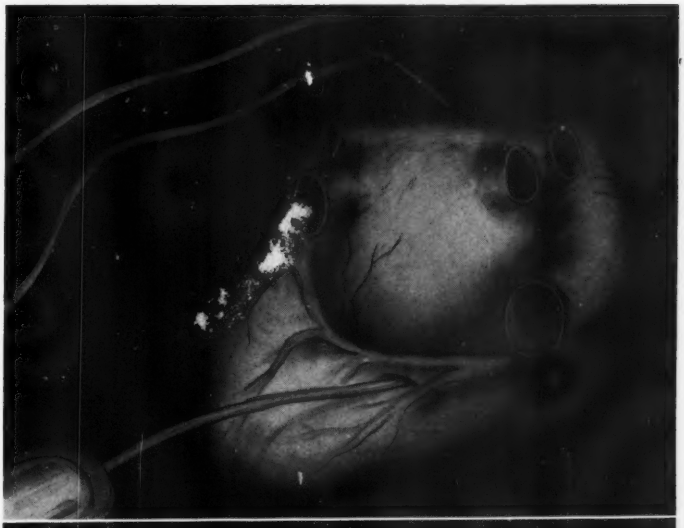


Fig. 4.

Fig. 5.

Fig. 4.—After the tip of the needle has entered the transverse sinus under digital guidance, the wire is then advanced so that the eye emerges anteriorly and can be easily threaded.

Fig. 5.—The pericardium-covered portion of the suture is drawn into the transverse sinus and beneath the circumflex coronary artery in the previously constructed tunnel.

Fig. 6.

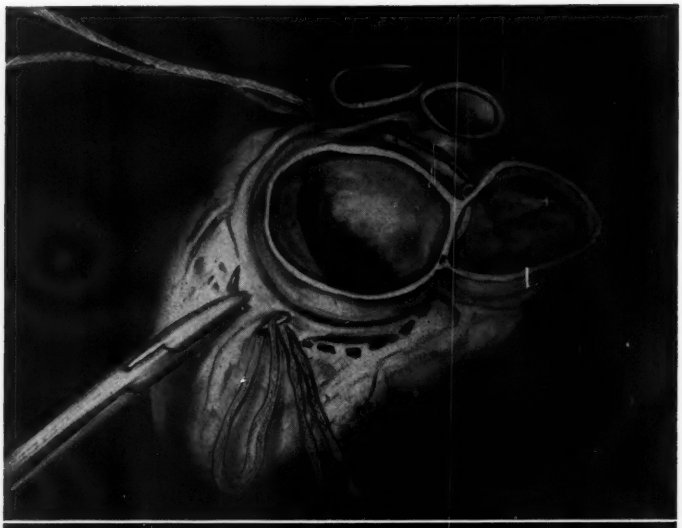


Fig. 7.

Fig. 6.—The posterior tail of the suture is then stitched into the atrioventricular fat pad beneath the circumflex coronary and cardiac vein and underpassing lateral descending branches of the circumflex. Coronary injury is easily avoided. The suture should lie within the fat pad and not in ventricular myocardium.

Fig. 7.—The suture completely placed and being tied anteriorly will lie concentrically over and just outside of the annulus. The suture is tied by the assistant as the operator palpates the regurgitant jet, the valve ring, and the valve leaflet action.

the passage of the suture (Fig. 5). Should there by any tendency to splitting of the thin myocardium in this area or should the emergence of the needle have been a little higher on the wall of the right atrium than desired, the tamponade and plugging effect of the sleeve of pericardium will effectively control any oozing. The anteromedial end of the circumferential suture is then drawn under the previously tunnelled circumflex coronary artery and the two ends now lie on the ventricular side of the mitral annulus. The posterior end of the suture is then brought forward to meet the anterior end by taking appropriate bites beneath the fat overlying the mitral annulus, taking care to avoid the left ventricular descending branches which arise from the circumflex coronary artery. Two or three such bites will accomplish approximation of the two ends of the circumferential suture (Fig. 6). At no point does the suture transfix myocardium except when entering and emerging from the right atrium.

The suture is then snugged in place by cinching down on the first throw of a square knot (Fig. 7). As this is done the suture eases into place in the V-like groove formed by the interdigitating muscle fibers of the atrium and ventricle and comes to rest exactly over the mitral annulus. The suture is further tightened, being guided in extent by the intra-atrial finger as it appreciates the diminishing degree of regurgitation resulting from coaptation of the valve leaflets. As a rule all regurgitation can be eliminated except a barely discernible whiff but this, of course, depends upon the pathologic status of the remaining valve tissues. The ring is usually reduced from a five-finger diameter to about a three-finger diameter and the effective valve orifice is not compromised. When maximum relief of regurgitation has been obtained, the square knot is completed and the mitral ring thereby fixed. The intra-atrial finger is then removed as the appendage is clamped. At this point, one frequently observes a diminution in the degree of left ventricular dilatation. What was formerly a large, distended, and incompressible left atrium is now frequently a flaccid, inelastic, easily compressible sac. Intracardiac pressures are repeated. The pressure in the left ventricle as a rule rises by 5 to 10 mm. Hg systolic and the pressures in the left atrium materially drop toward normal and not infrequently to within normal range. The original diffuse and intense systolic thrill felt everywhere over the left atrium is now either abolished or barely perceptible. The end diastolic pressure in the left atrium remains at a low level, indicating that no appreciable stenosis has been produced. In the average case, when the procedure has been properly performed, little, if any, cardiac irritability results other than an occasional ectopic ventricular beat. Frequently the cardiac rate, previously rapid, is now reduced to normal levels and is visibly more steady and effectual.

The appendage is ligated at its base, a portion of its tip amputated for histologic study, and the remainder oversewn. The operative field is flushed with warm saline, the pericardium partially closed and now, even though a portion of the pericardium has been resected, there is usually no difficulty in completely containing the heart loosely within the long, overstretched pericardium. A small opening is left in the pericardium to prevent

the entrapment of pericardial fluid. The lung is completely expanded, a lung biospy is obtained, the pleura drained by catheter, and the chest wall closed in the usual manner. The procedure will have been performed with little, if any, more trauma than is seen in the average mitral commissurotomy. Should appreciable stenosis have been present, an appropriate guillotine-type commissurotomy, either unilateral or bilateral, will be performed to free the leaflets from each other, thus ensuring maximum motion of valve tissues. The commissurotomy is best performed after the purse-string has been placed in its entirety but just before it is tied. As a rule, after commissurotomy, even though the intracardiac finger may now be unable to appreciate any increase in regurgitation, an interatrial pressure recording will almost routinely reveal that the pressure within the atrium has been very materially increased. This finding has proved to us over and over again that, in the presence of large amounts of regurgitation, intra-atrial finger estimation of the degree of regurgitation present is worthless. After commissurotomy the annular purse-string is tied down to its maximum degree as described. The entire procedure is monitored by a continuous oscillographic image and frequent recordings of the electrocardiogram.

SELECTION OF PATIENTS

Having developed an effective technique for the performance of total circumferential constriction of the mitral annulus and observed its effect in over 250 animal experiments over a period of two years, it logically followed that clinical application was in order. Ideally, from a purely surgical salesmanship viewpoint, it would have been highly desirable to employ the procedure on those patients exhibiting more minor degrees of mitral valve incompetence. A low operative mortality and a high percentage of living patients might attest the fact that the operation could be at least tolerated (improvement or no improvement), but was there justification at this point to subject patients in reasonable clinical health to the hazards of any new and intricate procedure of this magnitude? Under these circumstances would maximum objective data be forthcoming to ensure an honest and accurate appraisal of the procedure? We think not. On the contrary, it was felt that such application should not be employed until the efficacy of the procedure was entirely proved on the badly damaged rheumatic heart for, not infrequently, those procedures which seem to be highly effective in the laboratory prove to be less desirable or even detrimental to the human disease complex. Therefore, it was decided that initial clinical application would be employed only in those patients showing far-advanced, even terminal, degrees of rheumatic heart disease with mitral insufficiency. Thus, a number of patients previously denied surgery because of the hopeless nature of their cardiovalvular disability (primarily due to insufficiency) were recalled, the facts and status of the operation presented and then subjected to surgery as desired. It was determined that this pilot study should not include more than 25 to 30 patients. It was fully realized that due to the desperate condition of these

patients, perhaps few would eventually survive or be materially helped. Nevertheless the hope was ever present that some appreciable salvage might result.

ANALYSIS OF PATIENTS

Twenty-seven patients with rheumatic mitral insufficiency, pure or in combination with mitral stenosis, have undergone "mitral purse-string" to date. In 6, a concomitant commissurotomy was likewise performed and in 3 a commissurotomy had been performed one to two years before. The first mitral purse-string in the series was carried out on Jan. 20, 1955, and this patient is living a normal life today, sixteen months later. Twenty-four were female and 3 were male; the youngest was aged 7 and the oldest aged 52. The average age was thirty years.

Clinical Status.—Each of the 27 patients was in an advanced stage of chronic congestive failure (Stage IV). Fourteen were classified as intractable (terminal) failure (Group I), for despite the most diligent and heroic of medical regimens, their congestive failure could not be abolished and these patients came to surgery with variable degrees of pulmonary congestion, hepatomegaly, often with ascites and evidences of peripheral edema and stasis. Admittedly, patients in such a state of myocardial degeneration have long been recognized as the poorest of candidates for surgery with little hope of salvage. The remaining 13 patients (Group II) were likewise in an advanced state of myocardial degeneration and chronic congestive failure but by dint of tedious and prolonged medical therapy they came to surgery clinically free

TABLE I. CARDIAC CATHETERIZATION DATA
(PREOPERATIVE)

PATIENT	GROUP	RIGHT ATRIUM	RIGHT VENTRICLE	PULMONARY ARTERY	PULMONARY VENOUS CAPILLARY	ARTERIAL (BRACHIAL OR FEMORAL)
1. R. M.	II					125/75
2. B. F.	I	25/14	83/10	80/47	20/11	
3. P. F.	II		30/0	25/5	14/11	125/65
4. B. W.	I	15/7		58/30		
5. L. T.	I	8.5/2	75/-1	88/35		
6. R. S.	I	1.5/-3	20/-5	28/10	16/5	125/62
7. J. L.	I	1/-3	31/-6	30/11	16/-1	98/57
8. M. G.	I	7/0				
9. J. M.	I	10/6	80/2	76/36	29/15	
10. H. S.	I		48/0	46/11	49/17	
11. A. T.	II					
12. E. J.	II	3/-2	23/-4	20/4		130/60
13. G. S.	II	(9)	50/10	55/20	(16)	118/75
14. P. B.	I	5/-1	64/0			
15. K. K.	II	3/-4	12/1	12/5	10/6	
16. R. R.	I	4/-3	42/-3	45/18		
17. F. C.	II	5/0	35/-5	30/5	12/0	140/80
18. F. M.	II	9/4	38/7	38/18		118/69
19. G. B.	II	9/2	34/5	28/11	26/9	120/58
20. J. K.	I	10/6	35/5	35/17		140/48
21. J. S.	I	14/10	60/0	60/20	30/18	120/64
22. G. M.	II	-3/-10	35/-7	40/6	15/0	124/76
23. D. W.	I		60/0	60/18	40/12	120/80
24. E. H.	II	13/5	92/5	92/44	52/22	140/84
25. H. M.	I	24/15	84/10	80/40	52/22	126/80
26. M. L.	II	7/2	40/3	36/13	32/12	120/70
27. G. L.	II		38/7	37/18	30/12	120/60

of decompensation. The contrasting results obtained in these two groups of patients have been extremely enlightening and have served to corroborate further the impressions gained as to the indications for surgery in valvular disease over the years in our rather extensive experience with the rheumatic heart.

Preoperative Studies.—Each patient was studied in the usual routine cardiac fashion familiar to all, including routine blood work, blood chemistry, urinalysis, electrocardiograms, teleoroentgenograms and fluoroscopy, and phonocardiograms. In 25 of the patients a satisfactory cardiac catheterization was accomplished and these data have been summarized in Table I. It will be noted that in one half of the patients catheterized the pulmonary vascular and right heart pressures were two to four times higher than normal. Hypertension in the lesser circuit of this degree, while a common finding in mitral stenosis, is distinctly uncommon in the average case of mitral insufficiency.

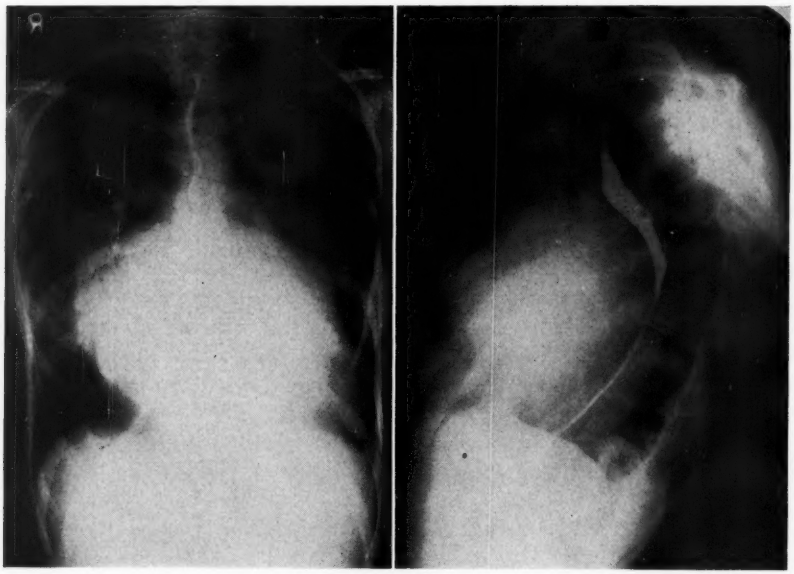


Fig. 8.—Posteroanterior and left lateral teleoroentgenograms of Case 5 which typify the findings in cases of Group I.

The degree of cardiac enlargement shown radiographically in each patient was marked. A typical example of this type of cardiac enlargement characteristic of the patient with mitral insufficiency in Group I of this series is shown in Fig. 8.

The changes in the electrocardiogram of each patient were extensive and most variable. This aspect of the findings in these patients has formed the subject of another communication to be presented shortly. It is sufficient to state here that, in 25 patients, atrial fibrillation was present and in the remainder

a normal sinus rhythm persisted. In 3 patients, an atrioventricular block was noted. Variable degrees of atrial hypertrophy and combined ventricular hypertrophy were routinely present.

Pre- and postoperative phonocardiograms routinely obtained confirmed the clinically obvious diminution in the mitral systolic murmur as shown in Fig. 11.

Group I—Intractable (Terminal) Failure.—There were 14 patients in this category. Five of these patients had undergone previous mitral valve exploration one to two years before, and in 2 of these a commissurotomy had been performed. Needless to say, in each of these 5 patients the performance of a "mitral purse-string" was doubly difficult for all intrapericardial landmarks were obliterated as were the left atrial appendages. The postoperative changes encountered were directly responsible for the operative death of one of these for an uncontrolled hemorrhage was produced while attempting to open the adhesion obliterated transverse sinus.

Seven of the 14 patients died within the first month and as such must be considered as operative deaths. In 2, ventricular fibrillation developed as the pericardium was being opened and these hearts could not be resuscitated. Two others went into ventricular fibrillation during the initial maneuvers of the passage of the purse-string. Cardiac massage and electric shock were unsuccessful in these until the purse-string was completely passed and tied rendering the mitral valve competent. Massage at this point effectively restored cardiac output and coronary filling so that defibrillation resulted. Despite this the patients were lost. The fifth patient tolerated the operative procedure surprisingly well despite her precarious condition only to succumb one week later to progressive irreversible heart failure. The remaining 2 died of operative hemorrhage, one for reasons alluded to above and the other as a result of a tear in the right atrium when the Davila needle emerged into the transverse sinus too high on the wall of the right atrium. These latter two deaths could well be classified as technical errors.

It must be apparent from the brief descriptions above that the majority of the deaths described were occasioned primarily by the far-advanced nature of the patient's disease. One can hardly blame the operation of circumferential annular constriction for the immediate effect of this procedure, hemodynamically speaking, was accomplished in but one of the 7.

The remaining 7 patients survived surgical intervention although in 2 ventricular fibrillation was present for twenty-four and sixty-five minutes, respectively. Both of these were restored to their preoperative rhythm only after the mitral valve had been made competent by circumferential constriction. One patient after rather considerable improvement in her congestive state during the first postoperative week shortly reverted to her previous intractable failure and died two weeks later. At post-mortem examination an extensive calcific aortic stenosis was noted. The presence of aortic stenosis had been recognized at the time of surgery but its degree of severity was misjudged so that this valve was not opened. Even had an aortic commissurotomy been considered, it is doubtful that the patient would have survived the added procedure. Two patients obtained rather remarkable relief from their

TABLE II. INTRACARDIAC PRESSURES BEFORE AND AFTER PURSE-STRING OBTAINED AT SURGERY

	INITIAL					AFTER COMMISSUROTOMY			AFTER ATRIOVENTRICULAR PURSE-STRING					
PATIENT	LEFT ATRIUM	LEFT VENTRICLE	AORTA	PULMONARY ARTERY	BLOOD PRESSURE	LEFT ATRIUM	LEFT VENTRICLE	BLOOD PRESSURE	LEFT ATRIUM	LEFT VENTRICLE	AORTA	PULMONARY	BLOOD PRESSURE	CLINICAL GROUP
†1. R. M.	30/10	108/-10							19/6	106/-3				II
2. B. F.	78/15	110/10		110/65	105/95				17/7	115/6		102/58	110/100	I
†3. P. F.	24/6	112/22							22/11	160/-5				II
4. B. W.	28/11	155/-2	130/65	54/25	130/15				18/6	150/-2	105/62	54/26	120/110	I
5. L. T.	38/5	102/-5	110/80	110/70	110/80				*					I
6. R. S.	27/7	80/-8		48/12	120/70				10/0.5	96/-4			110/70	I
7. J. L.	20/-3	90/-8		25/12	110/90				5/-4	92/-10		24/7	110/80	I
8. M. G.	48/13	128/0		120/60	120/80				25/7	112/0		94/44	140/80	I
9. J. M.	100/60	128/70		76/3	105/60				*					I
10. H. S.	34/14	115/0		33/10	90/80				*					I
11. A. T.	47/-10	100/-8		30/0	90/70				0/-10	100/-10		18/0	80/70	II
12. E. J.	48/8	160/0		48/16	180/20				15/10	140/0				II
†13. G. S.	29/7	138/-8	128/78	116/60	130/100				8/1	138/-2	132/64		130/100	II
14. P. B.	44/10	85/0	88/50	52/24	110/80	58/10			27/8	90/4	90/56	38/12	100/80	I
†15. K. K.	45/5	116/4		35/15	110/80				20/10	120/0		35/21		II
16. R. R.	*													I
†17. F. C.	30/2	110/-10		25/4	130/100				14/1	110/0		24/4	125/90	II
†18. F. M.	48/7	120/0			125/105				20/10	120/0				II
†19. G. B.	30/12	140/8	100/80		120/80				20/10	100/0			110/90	II
20. J. K.	35/10	110/-4	125/75			70/?	84/-8		*					I
21. J. S.	55/8	78/-4							*					I
†22. G. M.	35/5	100/-4			100/80				25/15	120/5			130/90	II
†23. D. W.	50/7	125/7			125/?				27/7	130/7			130/?	I
24. E. H.	60/18	120/7	124/84		115/70	92/18	100/18		40/20	120/18	120/84		110/70	II
25. H. M.	64/12								64/25					I
†26. M. L.	30/12	104/3	112/68		100/70				25/12	100/5	104/68		100/70	II
†27. G. L.	43/15	124/4	100/60		100/70	52/13	100/6	70/60	33/15	120/4	120/64		120/80	II

*Pressures not taken due to operative difficulties
†Living patients to date.

preoperative congestive states. Indeed, all signs of congestive failure disappeared and they were restored to a considerable degree of normal activity. Both of them died rather suddenly and in an entirely unexpected, unexplained manner, one at three months and one at five months. Both had been very active and one had just returned home from a shopping tour. Post-mortem examination in each failed to reveal any cause of death. The hearts of these patients were referred to us for examination and in each the purse-string suture was accurately and well placed with no evidence of either myocardial or coronary damage noted. Both valves had been restored to a high state of competency by the purse-string procedure. Three other patients likewise improved very dramatically for a matter of weeks and months only to revert again to a state of slowly progressive congestive failure. These 3 patients eventually died at six weeks, two months, and eight months, respectively. Post-mortem examinations were performed in 2 of these and again it was noted that the purse-string suture was properly and accurately placed and had accomplished very effective improvement in valvular competency. It is of interest that the patient in this group who lived the longest was the one who survived sixty-five minutes of almost continuous ventricular fibrillation. Constant and apparently very adequate massage was maintained throughout for little if any cerebral damage resulted. The final patient is alive today, three months after surgery, and is considerably improved. In the light of her 13 predecessors as just described it is altogether probable that she too will eventually succumb to intractable failure for her original cardiac status was quite as bad as that of the others.

It goes without saying that the results from a clinical standpoint were most disheartening. No appreciable salvage was obtained other than the fact that the survivors were made more comfortable for most of their remaining days. On the other hand a great deal of highly valuable hemodynamic and technical information was obtained which has gone far toward the establishment of this procedure as an effective means of controlling mitral insufficiency for patients in an earlier stage of this condition. The following points have been clarified. (1) Once the technique has been mastered, it can be accomplished under the most harassing circumstances and under conditions of almost impossible extremes of pathologic complication. It is tolerated amazingly well even in the most precarious of patients. (2) Post-mortem data are available in 11 of the 13 patients who have died. In each of these examined hearts, the purse-string suture had been accurately placed and lay directly over the mitral annulus. By simple laboratory test and by observation of these hearts on our pulse duplicator[21] which allows for direct vision of valves in action, the mitral valve in all but 2 cases was corrected by at least 80 per cent. In one of the two uncorrectable valves there was essentially no valve tissue present. Both the aortic and mitral leaflets were so small from contraction and induration that no coaptation could be obtained short of producing an almost complete stenosis of the mitral annulus (Case 25). The other (Case 9) had undergone commissurotomy elsewhere one year previously. At that time the aortic leaflet of the mitral valve had been split in its exact center rather than an opening made at the presumed anterolateral commissure. The patient

developed, as might be expected, a tremendous regurgitation and why he did not die immediately is a complete enigma for, when such is done experimentally, death is prompt. Nevertheless, this patient did survive only to become a complete invalid from constant congestive failure. The purse-string was passed without difficulty but because the continuity of the aortic leaflet had been destroyed, purse-stringing under these conditions could coapt the aortic leaflet to the mural leaflet but could not coapt the bisected halves of the aortic leaflet itself. This patient died from unrelievable mitral insufficiency.

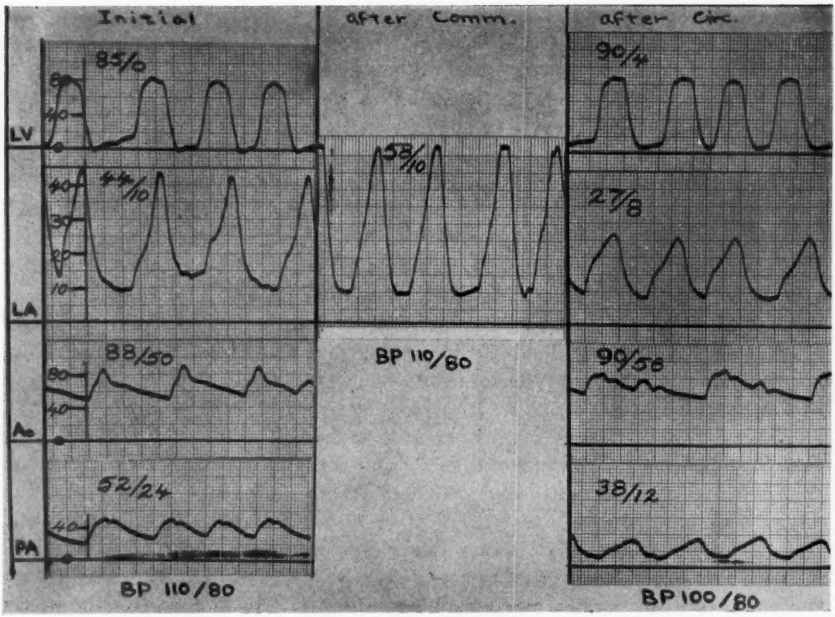


Fig. 9.—Pressure pulse tracings taken at surgery in a patient with mitral insufficiency and mitral stenosis (Case 14). In the initial tracings note the low ventricular and aortic pressure, the typical regurgitant contour and pressure range of the atrial tracing, and the elevated pulmonary artery pressure. Note that after commissurotomy there is a marked increase in the left atrial pressure. The regurgitant jet palpated by the exploring finger is not a reliable indication of whether regurgitation has been aggravated or improved by commissurotomy. After placement and tieing of the "purse-string," the ventricular and aortic pressures rise, the left atrial pressure falls, and the contour changes favorably, the pulmonary artery pressure falls. In most instances the change noted in pulmonary artery pressure may be small but in all the cases studied the left ventricular, aortic, and atrial changes are consistent.

(3) Proof of the above statements is shown in Table II. The reduction in left atrial pressure was highly satisfactory in all but the above-mentioned cases (Cases 9 and 25). Pressure curves in the left atrium likewise were restored to a more normal contour as were those obtained from the left ventricle (Fig. 9). (4) In no instance were the coronary arteries compromised, no erosion into the left atrium occurred, there were no appreciable myocardial changes and no stenosis was produced.

These data were essential to the proper continuation of this program.

Group II—Chronic Congestive Failure Clinically Correctable.—There were 13 patients in this category. One of these patients had undergone previous mitral valve exploration two years before but insufficiency and not stenosis had been found. Re-exploration of this patient at this time did not present the technical difficulties noted in the 5 similar cases in Group I.

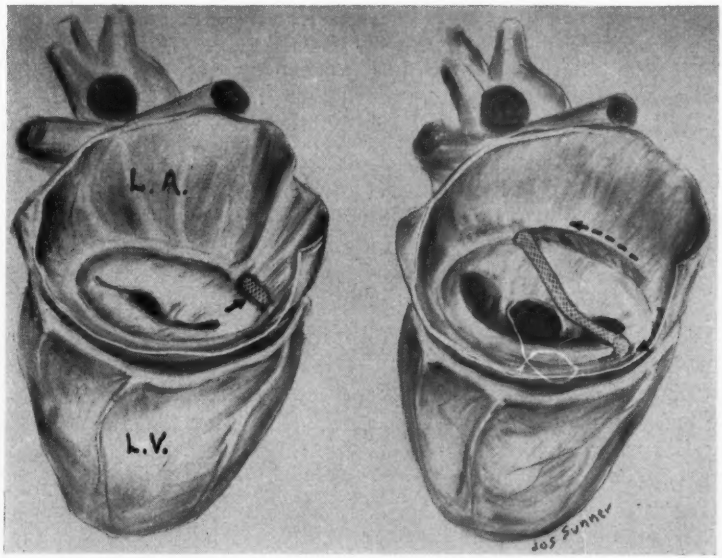


Fig. 10.—Drawing of the findings at autopsy in Case 11. The illustration on the left shows the position of the suture. Penetration into the lumen of the left atrium in this manner constitutes a serious technical error which can be avoided by careful palpation of the posterior commissural area of the ring after placement of the suture and withdrawal of it if it should be so placed. It is essential that in introducing the needle from the posterior aspect of the heart it be directed correctly into the right atrium. The figure on the right illustrates the erosion of the suture in two directions on the circumference of the ring beginning at the points of perforation (indicated by the dotted arrows). When the suture is properly placed, erosion does not occur.

Two of the 13 patients died within the first month and were considered operative deaths. One of these should have obtained a highly satisfactory result judging from the favorable nature of the valve leaflets and from the extremely satisfactory reduction in the left atrial pressure (Case 12). Inexcusably but inadvertently she received 500 c.c. of totally incompatible blood and passed away rather promptly after surgery from overwhelming intravascular hemolysis. The other patient was the second example in this series of a patient who, for all intents and purposes, had no remaining effective valve tissue. The aortic leaflet was but one centimeter in radius and most of the mural leaflet was nonexistent. At the posteromedial extremity of the mural leaflet there was a small amount of valve tissue which was attached to a short chordae tendineae that had long since been ruptured and this tiny portion of valve was flail-like. Even so, mitral purse-string did accomplish some reduction of valvular insufficiency although left atrial pressures still remained at

a much higher level than that seen in all the other cases in this group (Case 24). This patient died unexpectedly on the fourth postoperative day following a period of bradycardia. Post-mortem examination could establish no cause of death. One additional patient in this group died three months after surgery from subacute bacteral endocarditis. Post-mortem examination revealed that this patient had been the victim of the one truly technical error in the entire series. Fig. 10 illustrates that during the passage of the purse-string suture a tiny portion lay exposed within the lumen of the left atrium, at the posteromedial commissural area. As time passed this portion of the suture had cut well out into the lumen of the left atrium and thus lay like a strut across the lumen of the atrium. Extensive thrombosis had developed on this

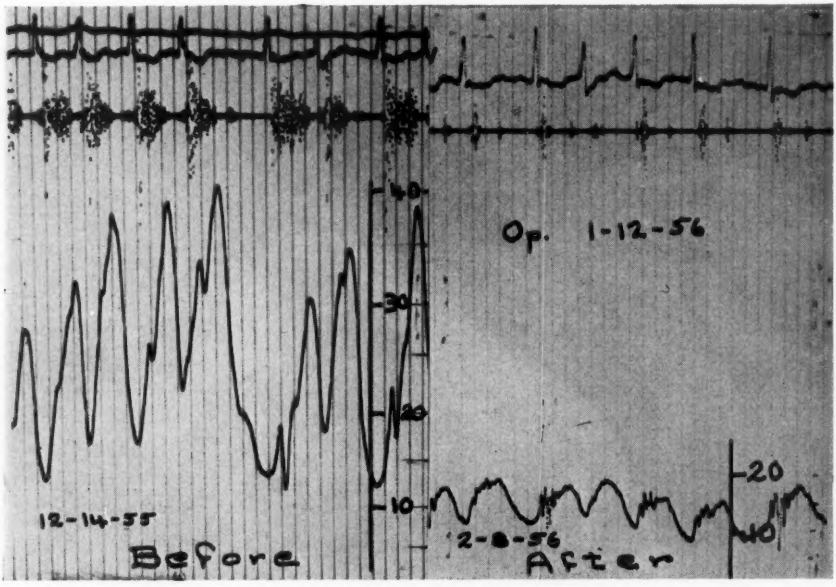


Fig. 11.—Left atrial pressure pulse recorded by transbronchial left atrial puncture approximately one month before and one month after operative correction of mitral insufficiency. Note the pre- and postoperative phonocardiogram as well as the atrial tracing (Case 18). (Courtesy of Dr. A. G. Morrow, National Heart Institute, Bethesda, Md.)

nidus leading to an uncontrollable blood stream infection. This case points up the extreme necessity for accurate placement of the purse-string for if a segment has been misplaced in the lumen of the left atrium, the result will be as just cited. This has been observed on a number of occasions during the early experimental animal work. In contrast to this, however, that portion of the suture which lies in the right atrium, because it is snugly against the interatrial septum and is not subject to the development of a cross luminal position, rapidly becomes endothelized and buried, a process which takes but two to three weeks. In all post-mortem specimens examined, this right atrial segment of the circumferential suture was buried in this fashion.

The remaining 10 patients are living to date. Two of these are sixteen months postoperative (Cases 1 and 3), 2 at eight months (Cases 13 and 15), one at six months (Case 17), 2 at five months (Cases 18 and 19), one at four months (Case 22), and 2 at two months (Cases 26 and 27). Those 5 patients who are now living six months to one and one-half years after surgery have obtained rather excellent results, are living relatively normal lives and are carrying out a full schedule of household activity. Much the same can be said for those under six months but not enough time has elapsed to make a definite statement to that effect.

Objective evidence of the correction of mitral insufficiency and the subsequent improvement of patients so treated is available in the form of pre- and postoperative phonocardiograms, teleoroentgenograms, and left atrial pressures taken before and after mitral purse-string at the time of surgery. As yet, none of these patients has been subjected to a formal postoperative cardiac catheterization.

An example of the type of pre- and postoperative phonocardiographic tracing routinely obtained is shown in Fig. 11.

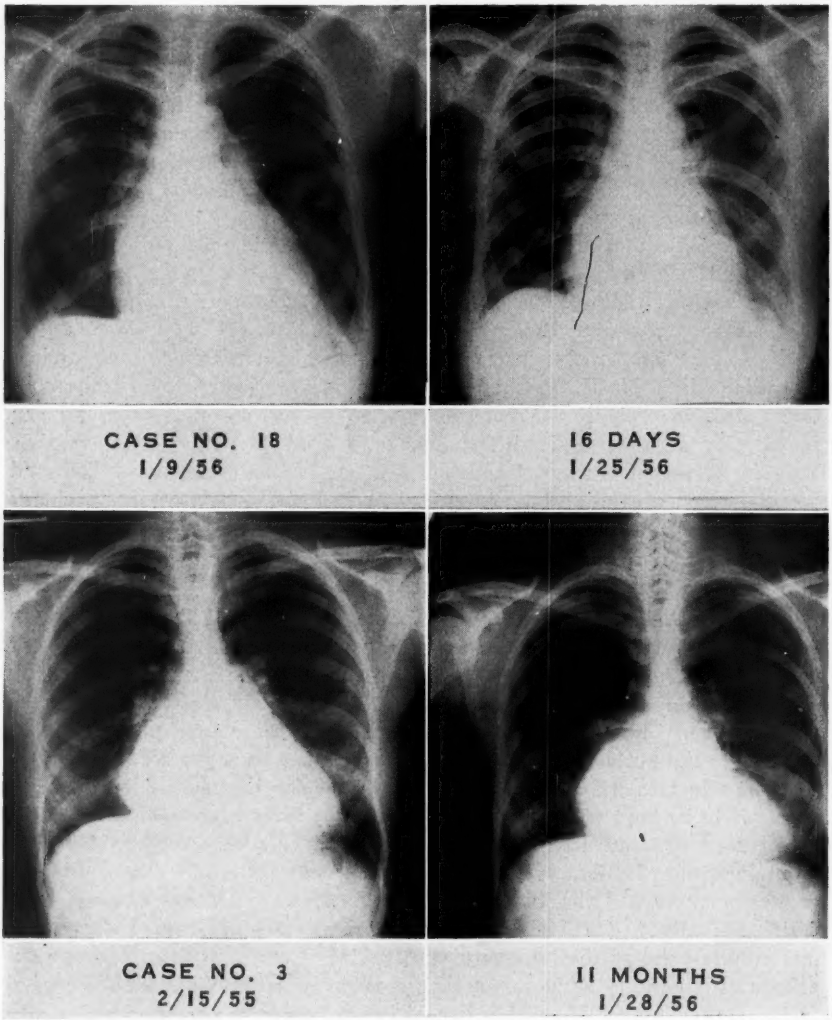
The obvious radiographic change in cardiac contour and diminution in size in the cardiac silhouette is reproduced in Fig. 12.

Pre- and postoperative purse-string pressure data within the left atrium are available in Table II. It will be noted that in most instances a highly satisfactory drop in pressure was obtained. The data in these living patients can conveniently be compared with similar data in those who did not survive. Examples of actual pressure tracings taken within the left atrium before and after annular constriction both in cases of relatively pure mitral insufficiency and in those complicated by concomitant mitral stenosis are shown in Figs. 11, 13, and 14.

COMMENT

An honest and sincere effort has been made to present the entire picture of our initial four-year effort to overcome surgically the detrimental effects of one of the commonest of valvular problems—mitral insufficiency. Much work and application lie ahead before one can categorically state that this procedure, or any other, materially affects the clinical course of patients so afflicted for the better. An attempt has been made to approach the problem in a most scientific and academic manner and the continuation of work on this project is to be kept at the same level. At least three aspects of the over-all project need constant attention. (1) Can the technique be further improved to obtain a more satisfactory hemodynamic effect by either closed or open heart surgery or will the final solution await the development of some form of artificial valve? (2) Are the patients, such as those in Group I whose ultimate results were so dismal, truly unsalvable? Continuing work in our experimental laboratory suggests another possible approach. Our laboratory associate, Dr. Domingos Moraes, is presently studying the effects of artificially produced chronic mitral insufficiency.[22] It is well known that the acute production of marked and significant mitral insufficiency in the laboratory animal is routinely attended by rapid death from fulminating pulmonary congestion.

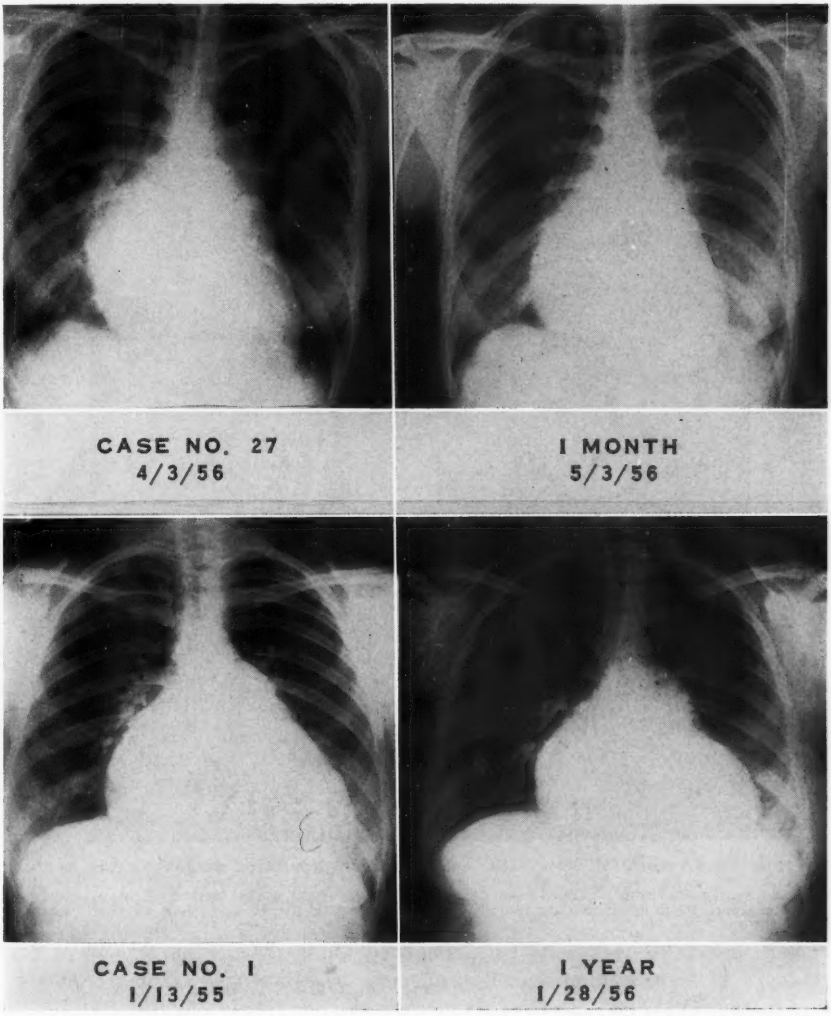
Therefore, to date no detailed reports of chronic mitral insufficiency are available in any quantity. Moraes has observed that when total ligation of the inferior vena cava below the renal veins is performed at the same operation as is the production of significant mitral insufficiency, approximately 50 per cent of the animals will survive. A considerable group of animals so prepared is presently being followed and the typical findings of clinical chronic mitral



A.

Fig. 12.—*A* and *B*, Posteroanterior teleoroentgenograms of 4 cases which typify the changes noted. The decrease in size of the left ventricle as well as the relief of tension in the left atrium, rendering it quite flaccid, is obvious immediately at operation. This diminution in cardiac size is evident on the early postoperative x-rays and persists or improves further in the late postoperative periods.

insufficiency are being observed. A possible solution applicable to patients in intractable congestive failure resulting from mitral insufficiency might be the preliminary ligation of the vena cava. Presumably by temporarily reducing the return of blood to the right heart and pulmonary vascular bed, such hearts may obtain a "breathing spell" and recover a measure of myocardial integrity. Mitral purse-string then performed at a more optimum time conceivably may be better tolerated and result in an improved clinical state. (3) The unexplained death in Cases 6 and 7 at three and five months, respectively, when



B.

Fig. 12, *B.* (*For legend see opposite page.*)

these patients were enjoying better health (free from cardiac congestion), than they had known in years, need a solution. Possibly Case 24, who died rather suddenly on the fourth postoperative day, falls into this same category although in this instance it was not possible to improve satisfactorily left atrial dynamics which of course might explain her unexpected death. Whereas in

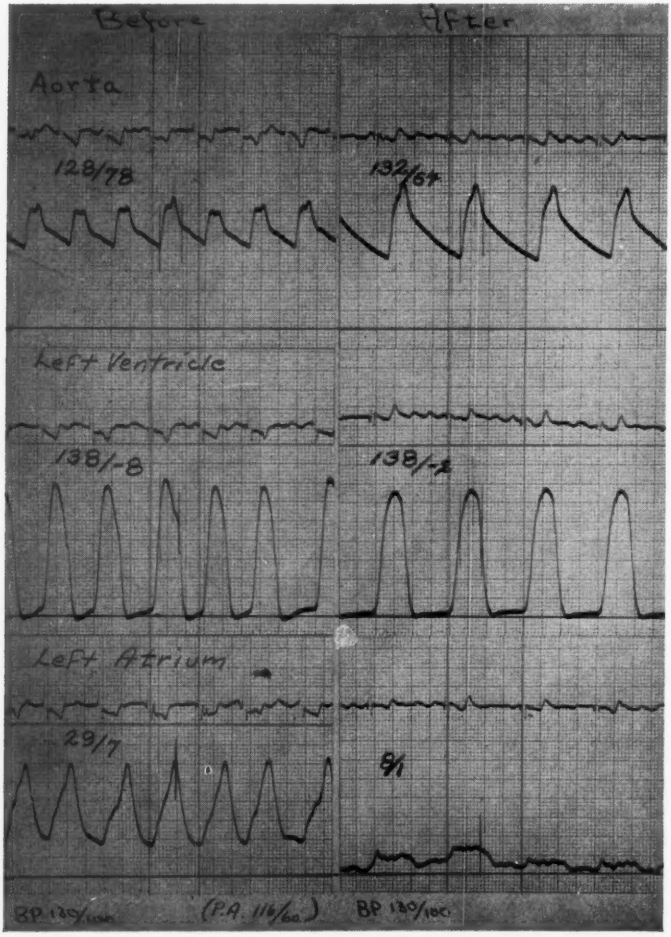


Fig. 13.—Tracings of aortic, left ventricular, and left atrial pressure pulses in Case 13, taken at surgery. In those recorded before the procedure, note the left ventricular contour in the ejection phase. Also note the typical regurgitant contour and pressure of the left atrial tracing. The changes in all three pressure pulses are ideal in this instance.

none of the experimental animals, followed for many months, was there any evidence of a conduction defect presumably occasioned by trauma or distortion to the bundle of His, possibly some such complication as this may be involved. Why this type of reaction should occur at such a late date is inexplicable. In this regard transient atrioventricular block was observed during

the postoperative period in 2 patients (Cases 17 and 19) but both cleared without sequelae. Case 11, who eventually died three months after surgery from subacute bacterial endocarditis, had an atrioventricular block both before and following surgery.

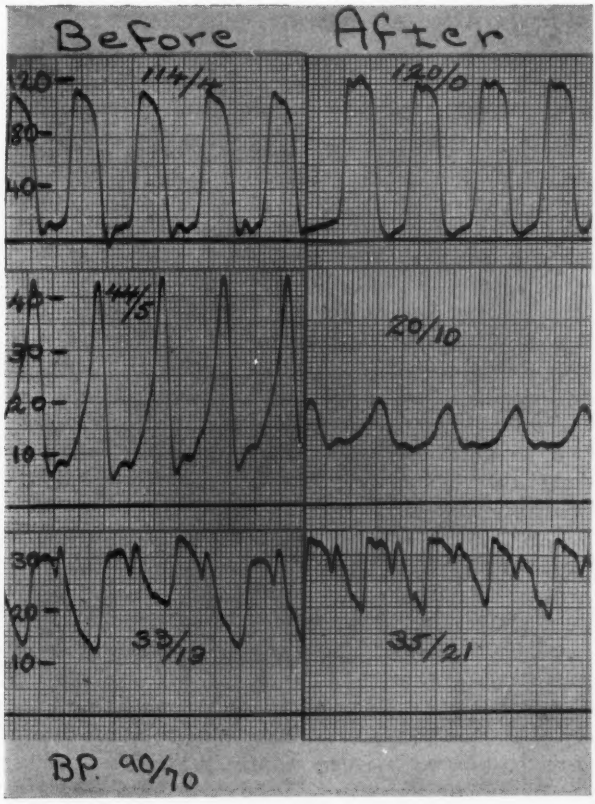


Fig. 14.—These tracings were taken at surgery of Case 15, a 7-year-old child with pure, massive mitral regurgitation. Note the very satisfactory improvement in left atrial pressure pulse contour and pressure. In this case, the question of rheumatic activity preoperatively led to the speculation that myocardial damage might preclude a good result. This patient, however, has done remarkably well.

SUMMARY AND CONCLUSIONS

1. The concept that mitral insufficiency is the result of three basic pathologic changes, namely, absolute loss of valve tissue, contraction and foreshortening of the chordae tendineae, and dilatation of the mitral annulus, was presented in detail.

2. For surgical correction of mitral insufficiency to be effective, each of these three factors must be taken into account.

3. Controlled circumferential constriction of the mitral annulus favorably affects each of these factors by reducing the size of the mitral orifice to be

occluded, by coapting the contracted and scarred but still pliable valve leaflets, and by providing relaxation to the stretched and distorted chordae tendineae.

4. Not only will total controlled circumferential constriction relieve in great measure existing mitral incompetence but, by permanent fixation of the atrioventricular annulus, it prevents further annular dilatation and subsequent progression of an insufficient state.

5. The technique for the proper performance of total annular constriction—mitral purse-string—has been detailed.

6. After three years of intensely applied experimental study during which time experimental mitral insufficiency, produced by every conceivable means, was proved to be correctable in routine manner, clinical application seemed justified.

7. Although ideally from a purely surgical viewpoint it would have been highly desirable to employ the procedure on those patients exhibiting more minor degrees of mitral valve incompetence in order to ensure a low operative mortality and a high percentage of living patients, this did not seem justified in the initial phase of clinical application. On the contrary, it was felt that early application should include only those patients showing far-advanced, even terminal, degrees of rheumatic heart disease with mitral insufficiency. Once the efficacy of the procedure was demonstrated on the rheumatic heart to simulate the findings observed on the experimental animal the operative procedure should then be applied at its proper level—the patient with early progressive mitral insufficiency.

8. A pilot group of 27 patients with rheumatic mitral insufficiency, pure or in combination with mitral stenosis, was subjected to mitral purse-string. In 6 a concomitant mitral commissurotomy was likewise performed.

9. Fourteen of these patients (Group I) were in intractable (terminal) failure with variable degrees of pulmonary congestion, hepatomegaly, ascites, and peripheral edema at the time of surgery. Seven of these patients died within one month, 6 within eight months and one is living to date at three months. Despite the fact that in most instances mitral insufficiency had probably been corrected to a marked degree, pre-existing irreversible myocardial, pulmonary, and hepatic changes proved too great for the mere correction of the valve lesion to overcome. The fact however that mitral purse-string could be accomplished with accuracy under these conditions, that mitral stenosis was not produced, that no coronary damage ensued, that no erosion of the suture into the lumen of the left atrium occurred, that no additional myocardial damage was produced, that the procedure was tolerated in patients in so precarious a state, and that left atrial hemodynamics could be corrected, was ample evidence that the procedure deserves considerable merit and further trial on better patients indicated.

10. Thirteen other patients (Group II) in chronic congestive failure but temporarily correctable by medical means prior to surgery were likewise subjected to mitral purse-string. Three of these patients died within three months and two of these were due to avoidable technical errors. The remaining 10 patients are living in a vastly improved state from two to sixteen

months postoperative. Hemodynamic, radiographic, and phonocardiographic objective evidence is available and has been presented to substantiate the obvious merit of the procedure.

11. Further clinical trial in better patients in an earlier stage of their disease complex is not only warranted but mandatory.

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(For Discussion, see page 117.)

MITRAL INSUFFICIENCY: TREATMENT BY POLAR CROSS-FUSION OF THE MITRAL ANNULUS FIBROSUS

HENRY T. NICHOLS, M.D. (BY INVITATION)
PHILADELPHIA, PA.

VARIOUS surgical attempts have been made in recent years to restore competence to the regurgitant mitral valve. These efforts have been directed in the main toward addition of tissue[1-5] or a prosthetic device[6-9] between the leaflets, suturing of one leaflet to the other,[10] distortion of the mitral annulus,[11-14] and elevation of the leaflets either by stretching,[15] cutting[11, 12] the chordae tendineae or insertion of a prosthetic beneath them.[8, 9] Each of these procedures represents an attempt to correct the pathologic entity or entities which to the respective reporter seemed to be responsible for the insufficiency. It is of interest to note that many of the basic principles and techniques being used today for correction of mitral insufficiency have been applied previously in attempts at the production of mitral stenosis in the animal laboratory.[16-18]

There are several pathologic entities such as congenital perforation of one or both leaflets, congenital cleft leaflets, and detachment of the chordae which give rise to mitral insufficiency, but which occur so infrequently that they deservedly have received little surgical attention.

There are three pathologic entities which now are generally accepted as being responsible for mitral insufficiency in the majority of cases: (1) retracted leaflets (Fig. 1, *A*), (2) dilated mitral annulus (Fig. 1, *B*), and (3) shortened chordae tendineae (Fig. 1, *C*). It is not uncommon to find all three to be present, and it may be difficult or impossible to determine which is predominant. Regardless of which of the three entities is present, absent, or predominant in a combination, failure of one leaflet to make contact with the other during systole always is evident.

The physiologic effect of mitral insufficiency is a reduction in the percentage of the left ventricular stroke output which enters the aorta. As the heart increases its rate in response to physical activity, its mechanical efficiency rapidly decreases in relation to the demand for increased aortic output because of the disproportionate reduction in the length of diastole (or left ventricular filling period) in the shortened cardiac cycle. This results in excessive fatigue which is relieved only by physical inactivity.

Since fatigue seems mild in contrast to the more dramatic episodes such as dyspnea, hemoptysis, and embolism which are seen in mitral stenosis, some physicians have been led erroneously to conclude that mitral insufficiency is

From the Department of Thoracic Surgery, Hahnemann Medical College and Hospital of Philadelphia, and The Bailey Thoracic Clinic, Philadelphia, Pa.

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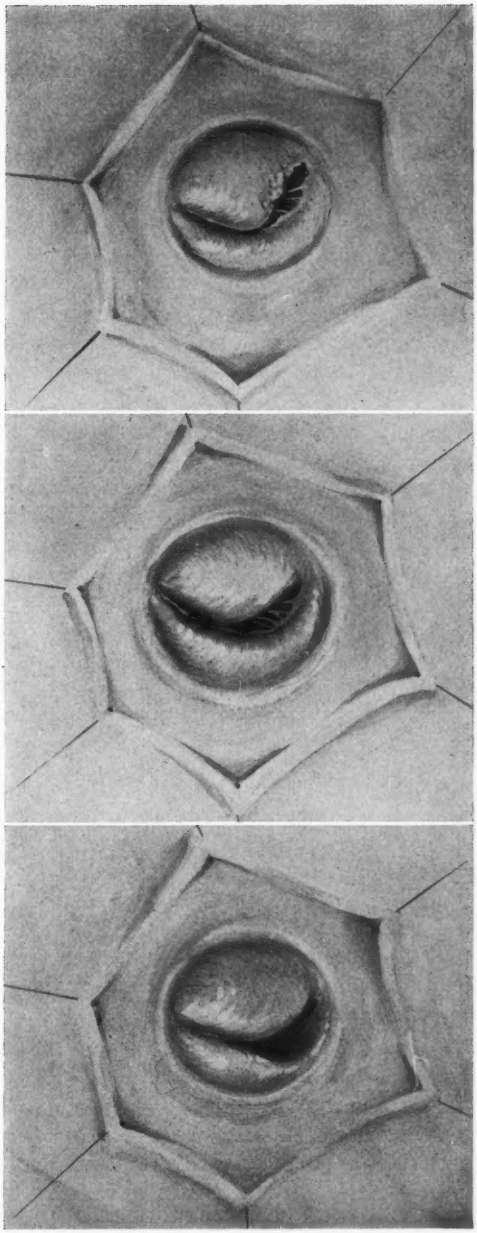


A.

B.

C.

Fig. 1.—*A*, Intrinsic shortening and retraction of leaflet margins is the commonest cause of mitral insufficiency. *B*, Dilatation of the mitral annulus fibrosus causes incompetence most marked in the region of the posteromedial pole of the valve. *C*, Shortened chordae tendineae draw the margin of a leaflet downward into the ventricular chamber and prevent systolic coaptation of the valve edges.

a benign condition. If the mitral insufficiency is great, the extra work load imposed upon the left ventricle eventually will lead to heart failure and death. Before this final event, the patient may be so restricted in physical activity that the joys of life seem few.

If the mitral insufficiency can be corrected, the mechanical efficiency of the left ventricle is increased and its over-all work load is decreased. The increased mechanical efficiency allows increased aortic outflow, especially during increased activity, lessens fatigue, and prevents or delays the onset of heart failure.

Attempts at introducing living tissue into the opening between the leaflets to block the regurgitation have resulted in a high operative mortality and frequently in disappointing end-results in the surviving patients. The application of prostheses into the same area likewise has eventuated unsatisfactorily.

These failures in attempts to block the opening between the leaflets have led to the development of various procedures designed to move one leaflet nearer the other thus enabling them to make contact during systole. Such operations have involved stretching and cutting shortened chordae to allow leaflets pulled down into the ventricle to rise into the atrium.[11-13] Prostheses have been placed beneath the mural leaflet without satisfaction.

We never have cut the chordae intentionally as we have been fearful of producing a flail leaflet. This, in itself, causes severe mitral insufficiency. Such a procedure would seem to have merit only in patients with shortened chordae and would seem contraindicated in those with retracted leaflets or dilated rings. Upward displacement of the mural leaflet has the same limitations in applicability.

Suturing of one leaflet to the other has been tried in an attempt to close the space between the two. We found that in some instances the leaflets were not strong enough to hold sutures under tension. Also, the inherent binding together of the edges of the leaflets by the pericardial sutures prevented descent of the edges into the ventricle and separation during diastole. If the leaflets were thin and pliable, this restriction was localized to the site of suturing. However, in those with thickened leaflets, the restraint was reflected throughout the leaflet edge and the entire cusp was limited in its movement with resultant stenosis.

More recently, our attention has been directed toward the annulus fibrosus and attempts have been made to displace one leaflet closer to the other so they might make contact during systole.

Hurwitt[19] suggested resection of portions of the annulus along with a wedge-shaped segment of ventricular myocardium. MacAllister[20] apparently was the first to attempt to constrict the mitral annulus fibrosus circumferentially in a clinical case. Davila and associates[13, 14] have applied a circumferential purse-string type suture around the entire mitral annulus. Borrie[21] has done the same procedure, apparently entirely independently. Kay and Cross[11]

have distorted the annulus in the region of the posteromedial commissure by placing sutures across the lumen of the atrium, through the annulus at either extremity, tying the free ends over buttons of cartilage outside the heart. Several modifications of this procedure to distort the annulus also have been made by Kay.[12]

The operation which we have named polar cross-fusion is also an anatomic alteration of the mitral annulus. In the development of this procedure, our objectives have been as follows: (1) to displace one leaflet closer to the other in the region of the incompetence so that their free margins may make contact during systole, (2) to place the approximating sutures around tissue that will hold under tension, (3) to avoid compromising normal leaflet action, (4) to avoid the production of physiologically significant mitral stenosis, and (5) to assure permanence of the anatomic alterations produced.

Our first attempt to accomplish the above objectives consisted of plication of the mitral annulus on the mural leaflet side by the placement of multiple interlocking mattress sutures within the substance of the annulus.[22] This shortened the annulus in this area and moved the mural leaflet closer to the septal leaflet sufficiently to correct regurgitations of small magnitude. However, it was not effective in abolishing the larger regurgitations. Also, this procedure was tedious and difficult technically because it required mobilization of the left circumflex coronary artery from its bed.

The above shortcomings led us next to consider plicating the annular ring by sutures placed between its mural and septal aspects in the region of the posteromedial commissure. In the animal laboratory, it was found that the sutures could be placed accurately and tied without great difficulty. Immediate and permanent fixation of the approximated portions of the ring was accomplished readily. We applied the polar cross-fusion technique to our first patient on Aug. 16, 1955, with survival.

TECHNIQUE

At the present time, the operation is performed through the left fifth intercostal space with the patient in the lateral position. If the fourth interspace is used, the exposure is limited for placement of the sutures. If the sixth intercostal space is used, there may be difficulty in relieving any coexisting mitral stenosis which might be present. The pericardium is opened by removing a 3 cm. wide strip of pericardium just posterior to and running parallel to the left phrenic nerve. This strip of pericardium is used to encase or ensheath the nonabsorbable suture to be placed inside the heart. The purse-stringed left atrial appendage is entered and exploration of the valve plus surgery for any existing mitral stenosis is carried out. The surgeon removes his right index finger from the atrium and moves to the opposite side of the operating table.

The ungloved left index finger is then introduced into the left atrium, the operator standing in front of the patient. A large curved needle swaged to a heavy Dacron suture is introduced through the left atrial wall at a point close to the incompetent valve pole (Fig. 2). If the valve is incompetent

posteriorly, the intracardiac tip of the bare left index finger is used as a guide to direct the point of the needle under the septal aspect of the annulus in the region of the posterior pole of the valve. The needle point then is redirected to continue across the atrial lumen and to pass just beneath the annulus on the mural leaflet side. Finally it emerges from the left ventricular wall close to the atrioventricular junction. The point of a second needle swaged to the opposite end of the suture then is engaged into the substance of the Dacron suture itself (Fig. 2) near its entrance puncture through the left atrial wall

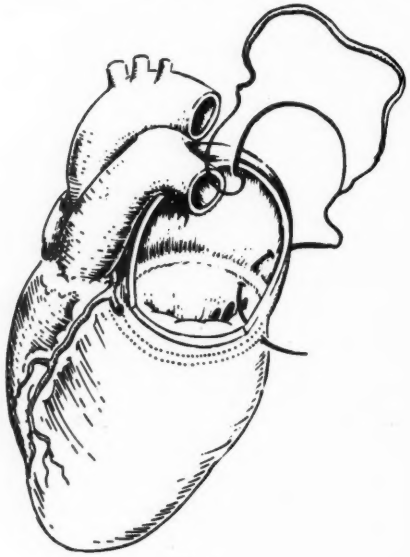


Fig. 2.

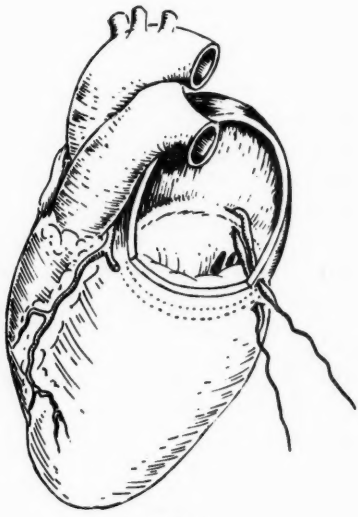


Fig. 3.

Fig. 2.—Placement of the plicating suture. One needle has pierced the left atrial wall, has been guided under the septal portion of the annulus fibrosus, has traversed the atrial lumen, and has passed under the mural portion of the ring to emerge from the left ventricular wall. The other needle point is imbedded temporarily within the Dacron suture in order to enter the atrial chamber through the same puncture. The pericardial sheath has been affixed to the mid-portion of the suture.

Fig. 3.—After the second needle enters the atrial chamber, it is caused to emerge close to the first but at a level just above (superficial to) the mural portion of the annulus.

in order to assure that it will pass into the left atrium through the same opening. Entrance through the same hole is important because it obviates the inadvertent inclusion of soft tissue within the loop of the suture when it is tied down. Necrosis of such included muscular tissue under pressure of the suture will tend to permit the approximated areas of the annulus to separate during the postoperative period.

After the point of the second needle is passed through the same atrial puncture site, it is disengaged from the Dacron suture merely by applying traction to the end of the suture which already has been passed through the heart. The needle point then is directed across the lumen of the left atrium to pass just above the annulus on the mural leaflet side and then to emerge from the left ventricular wall in close proximity to the other. The suture then

is adjusted, by traction on its ends, so that the part remaining within the heart is completely encased by the homograft pericardium (Fig. 3). The distal ends of the pericardial strip are clamped and then freed from the Dacron by cutting the attaching arterial silk stitches. The mobilized suture then is tied outside the heart, making certain, by use of the left index finger within the heart, that the two points of the annulus enclosed within the loop of the suture are brought into firm apposition (Fig. 4). Since there usually is a small amount of ventricular muscle caught within the loop of the suture between the knot and the underlying mural part of the ring, we have waited several minutes after making the first throw in the knot to allow such muscle to give way under the tension of the suture. The first throw is then tightened again, and this is followed by completion of several additional throws in the knot. A fine cotton suture passed through the substance of the Dacron tends to ensure

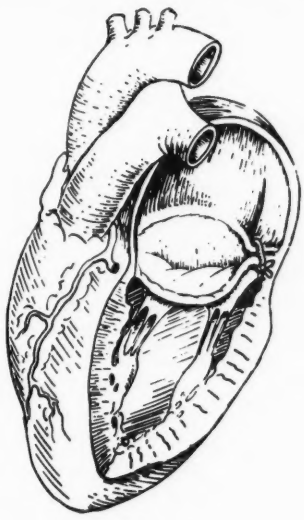


Fig. 4.

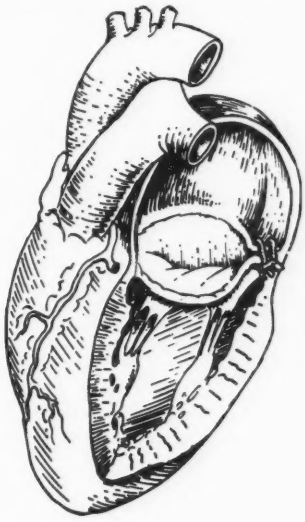


Fig. 5.

Fig. 4.—The suture is tied down as described in the text after the pericardial sheath has been separated from the Dacron in the region of the knot. At this point competence is restored to the valve in most instances.

Fig. 5.—In some individuals a second such plicating suture is necessary in order to overcome the entire insufficiency.

tightness of the knot. The emerging ends of the pericardial strip are approximated or tied together and this apposition is rendered permanent by placing several cotton stitches through them.

A second suture is placed alongside the first (Fig. 5), if it is necessary for the complete abolition of the regurgitant jet. At the present time, we are uncertain whether a second suture should not always be placed to act as a buffer to decrease the tension on the first one.

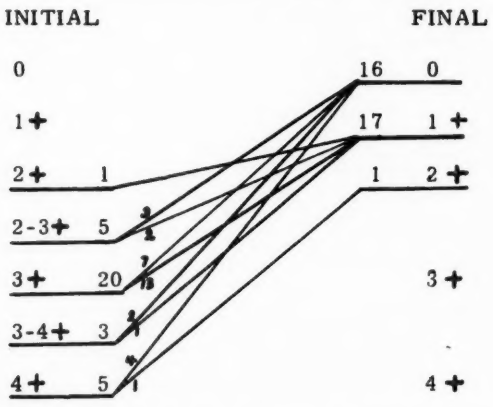
Cross-fusion of the annulus at the anterior pole of the valve can be selectively carried out just as is described for application at the posterior commissure in those few patients in whom the leak lies anteriorly.

DISCUSSION

The effectiveness of this procedure in blocking the regurgitation at the time of surgery has been most dramatic (Table I). Among the 34 patients operated on, the regurgitation no longer was palpable and apparently was abolished completely in sixteen instances. In 17 patients, there remained a palpable regurgitant jet which had been reduced to an estimated 1 plus magnitude. Many of these were just barely palpable to the ungloved finger, but if any regurgitation at all could be felt, it was recorded as of 1 plus magnitude. In only one patient was a 2 plus regurgitation permitted to remain. This was an extremely ill woman in whom one suture reduced the insufficiency from 4 plus to 2 plus, and it was felt best not to attempt placement of a second suture because of her extremely poor condition on the operating table. In the majority of these individuals, effectiveness of the correction at the time of surgery was evidenced by the lowering of tension within the left atrium and, in some, this was further attested to by a sudden and obvious decrease in left ventricular size.

TABLE I

ESTIMATED M. I. AT SURGERY



We have been encouraged to feel that we are accomplishing the objectives previously laid down. The bases of the leaflets certainly are brought into closer proximity so that their free margins are enabled to make contact during systole, thus effectively blocking the regurgitation.

The mitral annulus fibrosus seems to satisfy the requirement of a substance strong enough to hold sutures under tension. At least it is the only tendonlike structure available. The atrial wall and ventricular wall are muscular, and the incapacity of muscle tissue in holding sutures under tension is well recognized. The leaflets themselves will not hold sutures unless heavily fibrosed and, if such fibrosis is present, the creation of mitral stenosis is the price of correcting the regurgitation by this method. Polar cross-fusion of the annulus does not compromise leaflet action because only the leaflet's anchorage

point, the mitral annulus, is enclosed within the loop of the suture. The leaflets themselves remain capable of swinging with the alternations of the hydrodynamic stresses during the cardiac cycle. Physiologic mitral stenosis is not produced because the cross-sectional area of the atrioventricular passageway at the valve annulus is considerably greater than at the leaflet orifice. Permanent fusion of the plicated portions of the annulus should be assured by the use of the heavy nonabsorbable suture (80-pound test braided Dacron) plus the ensheathment of autogenous pericardium. The Dacron suture is used primarily for its bulk rather than its tensile strength in order to obviate any tendency of the suture to cut through substance of the annulus. The pericardial sheath around the suture likewise buffers any tendency toward cutting through. Ultimate conversion of the autogenous pericardium into dense scar tissue assures a fibrous coupling of the approximated areas of annulus (Figs. 6*A* and 6*B*).

The mortality (Table II) experienced by the first 34 patients operated on was gratifyingly lower than that of some of the procedures which we have performed in the past and some which have been and presently are being done by

TABLE II

Total cases (polar cross-fusion)	34
Concomitant mitral commissurotomy	9
Associated A. S., A. I. (aortic lesion not corrected)	2
Deaths to date	5 (14.7%)

others. The very fact that no deaths have occurred on the operating table has convinced us that the procedure is far safer than others we have tried. We have had to treat fibrillation once during surgery, correction being accomplished after the plicating suture was tied down.

The causes of death (Table III) lead us to believe that the mortality can be lowered in the future. Certainly the technical error is avoidable, and the basal skull fracture was not related to the operative procedure. The operation

TABLE III. CAUSES OF DEATH

1. Technical error (occlusion of right coronary artery)
2. Improper selection (incorrect evaluation of co-existent aortic stenosis)
3. Severe hypotension (during thoracotomy)
4. Basal skull fracture due to fall (33 days postop.)
5. Acute bacterial endocarditis

was completed in the patient with severe hypotension only because we felt that to retreat without giving mechanical relief to the heart assuredly would lead to fatality. Resistant strains of bacteria seem to be on the increase, but fortunately newer antibiotics also continue to appear. Selection of patients can, in some instances, only be settled by the philosophy of the patient, cardiologist, and surgeon in undertaking calculated risks.

Any attempt at evaluation of the results of surgery with reference to the level of physical activity would not be valid at this time. None of the patients as yet is one year postoperative. The majority of the patients have been

Fig. 6*A*.—Photograph of a mitral valve specimen showing a pericardial hammock which had been placed three years previously.

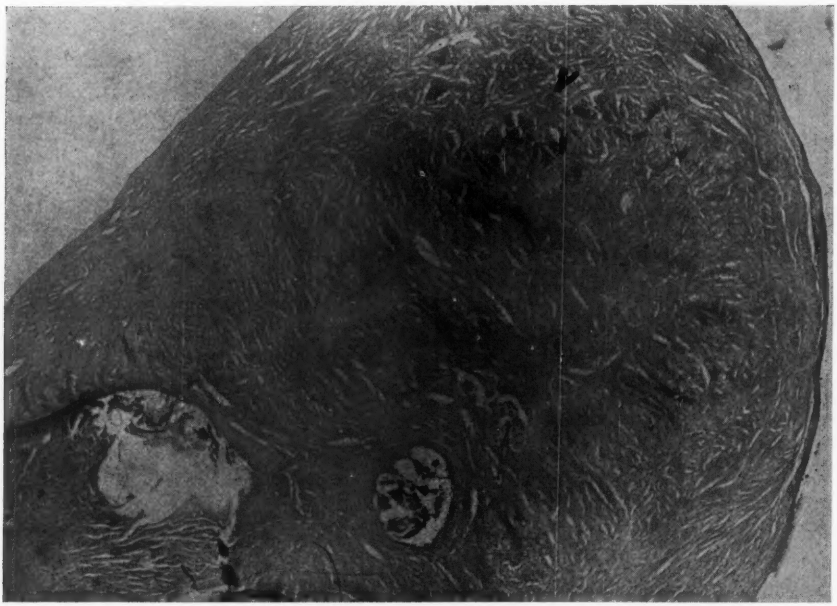


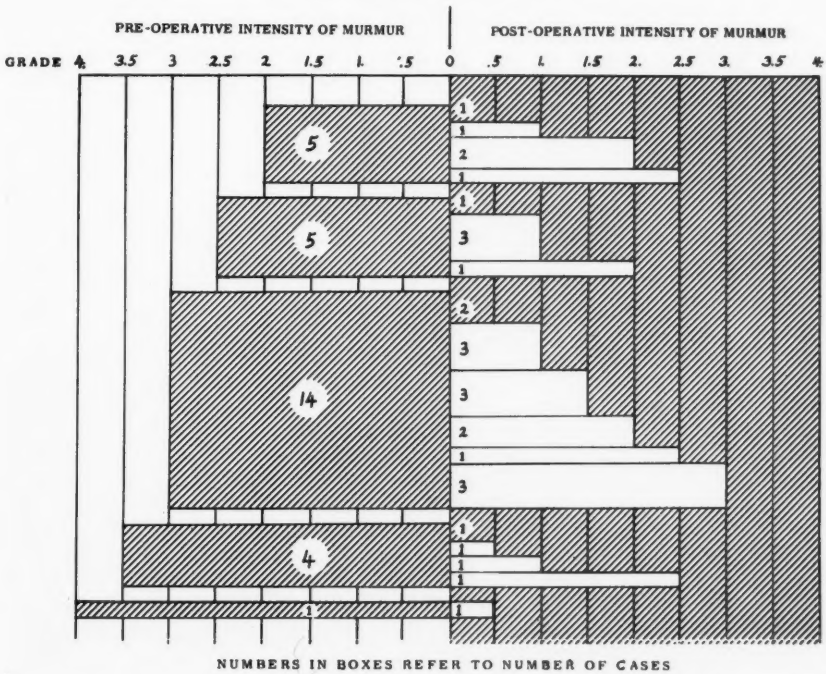
Fig. 6*B*.—Photomicrograph of cross-section through fibrosed pericardial hammock showing collagenous metaplasia.

operated on within the past five months, and have not, as yet, tested their maximal exertional ability. An evaluation of their status will be reported at a later date.

The fact that there is uncertain correlation between the intensity of a systolic murmur and the estimated amount of mitral regurgitation found at surgery is appreciated universally. The fact that no regurgitation is found in some patients with systolic murmurs, and that, in other patients, regurgitation is found, when no murmur was detected preoperatively, serves to demonstrate the impossibility of a true auscultatory correlation. However, we felt that a comparison of preoperative and postoperative murmurs might be of some usefulness and such is, therefore, presented in Table IV. Fig. 7 shows phonocardiographic studies in a patient in whom the systolic murmur was completely abolished.

TABLE IV

CHANGES IN SYSTOLIC MURMUR



NUMBERS IN BOXES REFER TO NUMBER OF CASES

We have performed left heart catheterization by the method of Björk[22] and Kent and associates[23] in 21 patients preoperatively and, to date, in 8 cases postoperatively. In none of the postoperative tracings has there been evidence that mitral stenosis was produced by surgery. The typical findings in the left atrial curves have been similar to those shown in Fig. 8 in which the enlarged V wave has been altered markedly and in which the mean atrial pressure has been reduced.

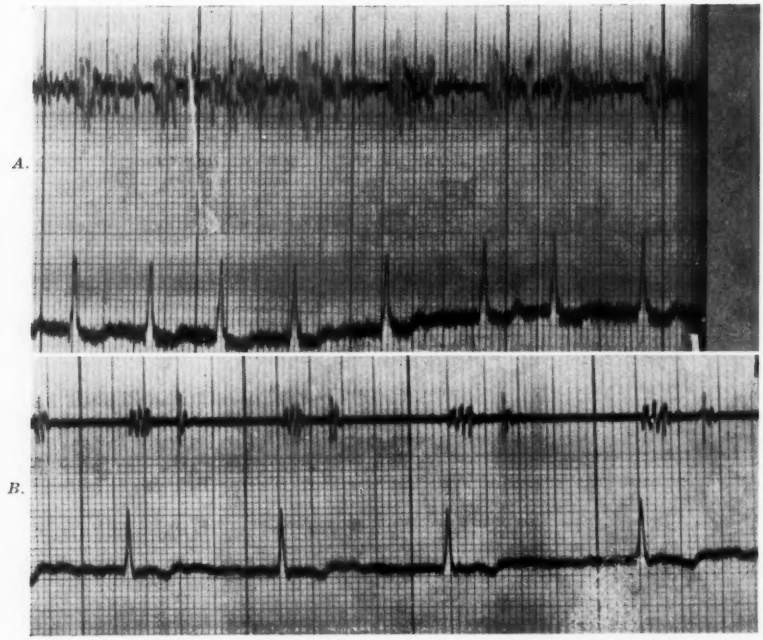


Fig. 7.—*A*, Preoperative phonocardiographic tracing indicative of long loud systolic murmur.

B, Postoperative phonocardiographic tracing in the same patient showing practically complete abolition of systolic murmur.

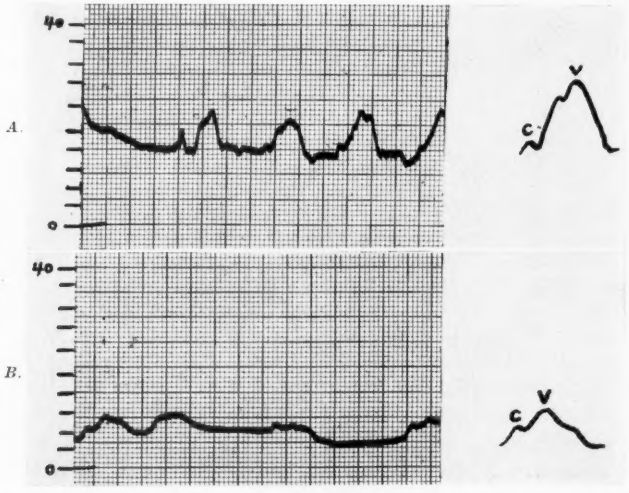


Fig. 8.—*A*, Preoperative pressure tracing obtained from the left atrium during left heart catheterization (Björk method). Note the large V wave suggestive of mitral insufficiency.

B, Postoperative pressure tracing obtained from the same patient showing normal C-V wave.

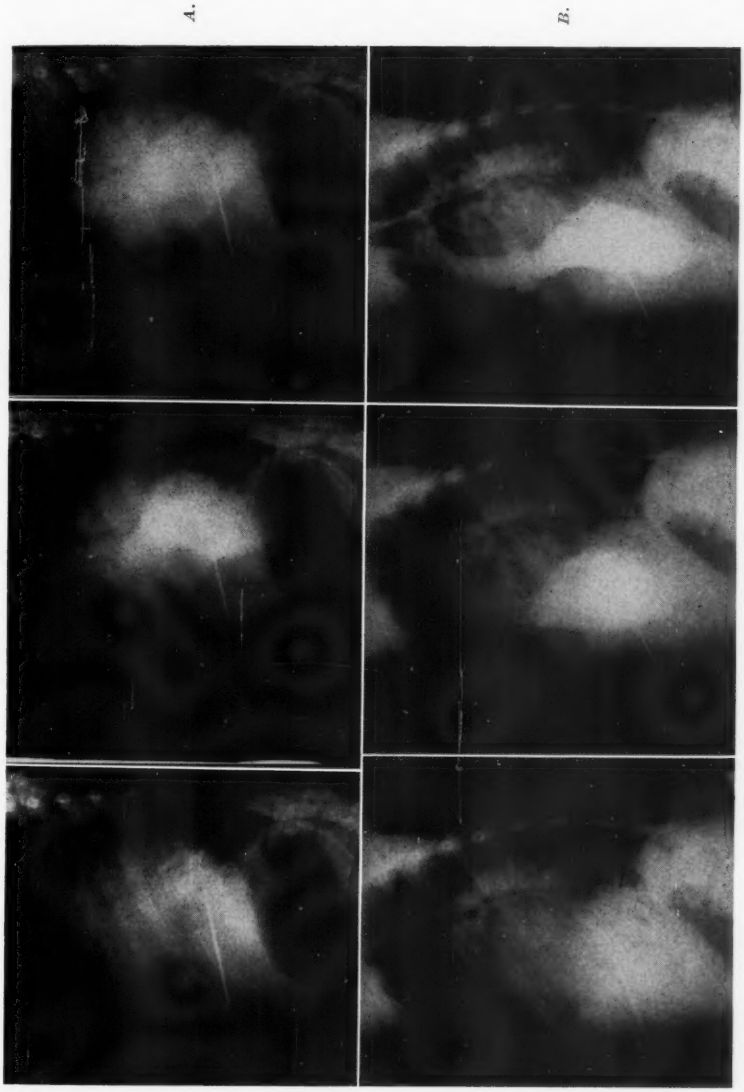
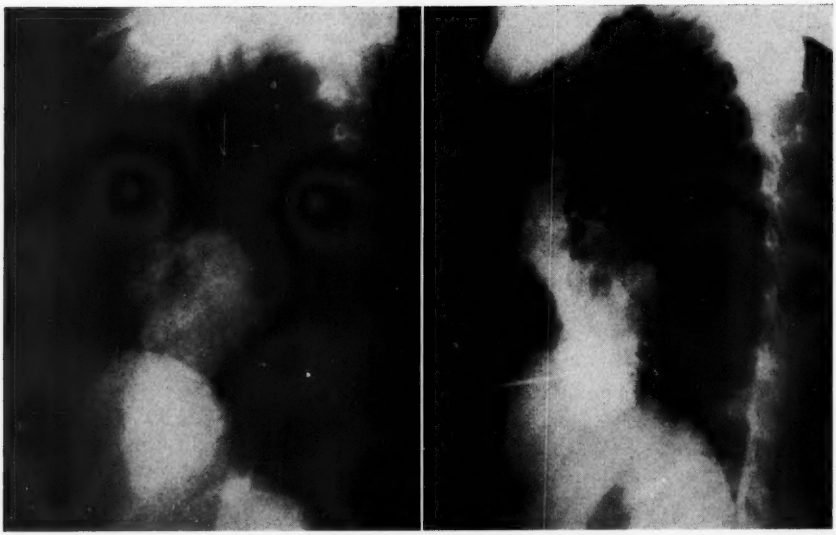


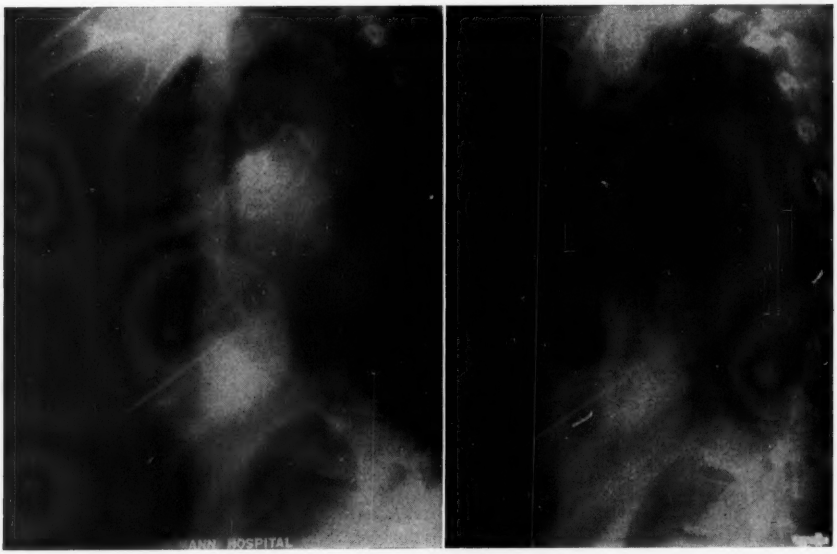
Fig. 9.—*A*, E. F., Serial radiograms taken respectively (left-to-right, April 10, 1956) just before, during, and immediately after direct injection of 50 c.c. of 70 per cent Diodrast into the left ventricular chamber (Smith technique). Note opacification of the left atrium indicating regurgitation.
B, Similar ventriculograms taken in the same patient three weeks (May 2 1956) after polar cross-fusion of the annulus. Note marked reduction in the extent of opacification of the left atrium.



A. *B.*

Fig. 10.—*A*, Preoperative ventriculogram obtained on Patient J. A., a 21-year-old white woman. Note opacification of the left atrium indicating an extremely incompetent mitral valve.

B, Ventriculogram obtained on J. A. three weeks after performance of polar cross-fusion of the annulus for a 3 plus incompetence of the posteromedial pole of the valve. Note the great reduction of opacification of the left atrium which is indicative of a similar reduction in the mitral insufficiency.



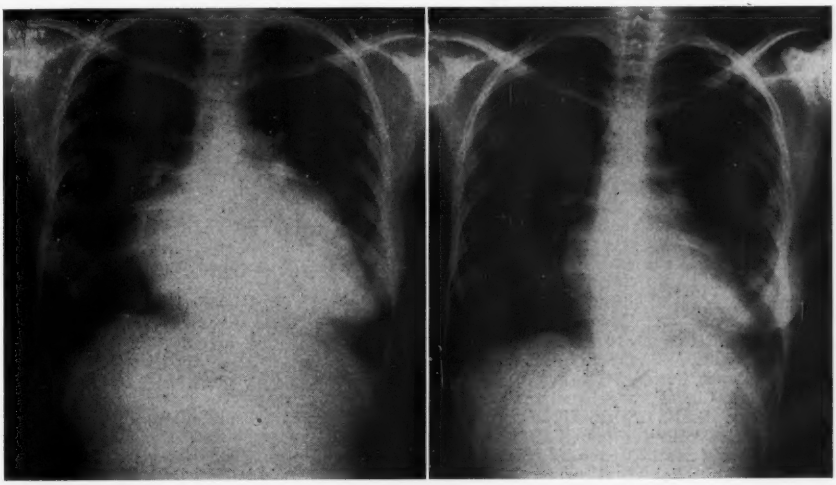
A. *B.*

Fig. 11.—*A*, Preoperative ventriculogram obtained on Patient M. S., a 21-year-old white woman. Note extreme opacification of left atrium.

B, Ventriculogram obtained on the same patient two and one-half weeks after polar cross-fusion of the posteromedial pole of the valve for a 3 plus insufficiency. Note the near-absence of opacification of the left atrium indicating the great degree of restoration of mitral competence which has been achieved.

Left cardiac ventriculogram by direct needle puncture into the left ventricle as advocated by Beato Nuñez,[24] and Smith and associates[26-28] has been performed in 19 patients preoperatively, and in 6 postoperatively. Some of the postoperative ventriculograms have shown no demonstrable dye entering the left atrium, while the others have shown a decreased amount in comparison with the preoperative ventriculogram. Figs. 9, 10, and 11 demonstrate comparative pre- and postoperative ventriculograms.

Decrease in heart size has been demonstrated in some of the postoperative patients. Fig. 12 shows such decrease. Others have shown slight reduction in size. No alteration has been seen as yet in the remainder. To evaluate the ultimate changes in heart size will require a longer follow-up period.



A. *B.*

Fig. 12.—*A*, Preoperative 72-inch posteroanterior roentgenogram of L. S., a 31-year-old white woman with severe (3 plus) mitral insufficiency.

B, Similar roentgenogram obtained 3 weeks after polar cross-fusion of the mitral valve. The great reduction in heart size suggests improvement in cardiac function.

SUMMARY AND CONCLUSIONS

The surgical technique for establishing polar cross-fusion in mitral insufficiency has been presented. The objectives of the operation and the methods of accomplishing them are presented. The operation has been used successfully in valves showing the three most common pathologic deformities, shortened or retracted leaflet, dilated ring, and shortened chordae tendineae. We have used it in 2 cases in which there were no palpable chordae attached to the mural leaflet. This lack of support permitted this leaflet to override the septal leaflet.

In one of our 34 patients, the procedure was applied across the anterior pole of the valve only, as this was the region of predominant regurgitation.

In another, it was applied across the area of the anterolateral commissure in addition to the posteromedial commissural area. In the remaining patients, the procedure was carried out only across the posterior pole of the valve where the vast majority of insufficiencies occur.

Obviously, polar cross-fusion of the annulus cannot be helpful in an extremely rigid or calcified valve.

We have no experience with the procedure in repairing insufficiency created surgically during the performance of mitral commissurotomy. In cases in which the chordae have been severed or the leaflets inadverently torn or cut, it is doubtful if it will be of as great value as in those lesions which are produced by the rheumatic process. It may well prove of value, however, in those instances in which the division of the commissure has been done accurately with respect to the leaflets but in which the incision has been allowed to continue on through the mitral ring. It may be possible in these individuals to restore the ring integrity by polar cross-fusion.

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DISCUSSION

(PAPERS BY GLOVER, AND DAVILA; AND NICHOLS)

DR. MELVIN NEWMAN, Brooklyn, N. Y.—Our group at the State University of New York in Brooklyn believe that the operation of the future will be the complete replacement of the mitral valve by a plastic prosthesis.

(slide) This simple ball valve has been used for complete replacement of the mitral valve in 25 dogs. The features of the operative technique are first, complete by-pass of the circulation by a pump oxygenator; second, ventricular fibrillation produced by electric shock, and this completely obviates the problem of air embolism; and third, insertion and fixation of the valve under direct vision through a left atrial approach. Ventricular fibrillation has been promptly reversible in all dogs and in the 2 patients on whom operative correction of interventricular septal defects has been done. The ball valve, as shown, has permitted survival for a maximum of four months and is unsuitable for clinical use because of thrombus formation, emboli, imperfections in fixation technique. However, the operative approach can be applied to any type of mitral valve surgery which may be done in the future.

DR. HARRISON BLACK, Boston, Mass.—I have enjoyed very much the presentations of Drs. Glover and Nichols, and their important contributions to this subject. On Dr. Harken's services in Boston we have wrestled with this difficult problem over the years. It is apparent that, although some patients have been greatly helped, the ultimate salvage when they are followed for a significant time leaves one dissatisfied with all the techniques currently available including intra-auricular baffles, annulus distortion, external annulus constriction, and external bolstering of the posterior herniation of the valve complex. At the risk of seeming repetitious, however, I believe I should re-emphasize several principles which constitute a distillate of our experience in dealing with this problem.

First of all, as we have been reminded by the authors, it is extremely important not to immobilize the leaflets. Any procedure which does this, as we have stressed on numerous occasions in the past, is going to result in damaging obstruction to forward flow. We cannot trade stenosis for insufficiency.

Second, the aneurysmal dilatation of the left auricle which occurs in the advanced stages of this disease must be corrected. Patients with predominant mitral insufficiency do not become significantly symptomatic, for the most part, until an aneurysmal dilatation of the left auricle has occurred and left ventricular failure has supervened. As the systolic jet weakens the auricular wall the chamber enlarges to enormous size and the pull of gravity in the erect position causes it to fall posteriorly toward the diaphragm. This in turn results in a sliding hernia of the valve leaflets at the posterior commissure which aggravates their incompetence by pulling them further apart. It seems reasonable

to assume that this particular anatomic situation accounts for the concentration of most significant mitral insufficiency about the posterior commissure. Therefore, any technique for the correction of mitral insufficiency, in addition to reducing the circumference of the annulus and preserving leaflet mobility, must take into consideration this third dimensional concept by reducing the herniation and possibly also by plicating the redundant auricular wall.

Finally, in evaluating the results of any procedure for the correction of mitral insufficiency we must limit ourselves to pure cases. It is, of course, obvious that no one can choose to operate only on pure incompetence since many mixed cases are invariably encountered. In assessing results, however, it is well to recall that Ellis* in analyzing the outcome of valvuloplasty in the first 500 of our cases of predominant mitral stenosis, found on long-term follow-up that even when marked insufficiency was found at operation, the correction of the existing stenosis alone resulted in improvement in 50 per cent of the cases. When moderate degrees of incompetence were encountered, 70 per cent showed improvement after correction of the stenosis alone.

As concerns the evaluation of what one has accomplished immediately, the reduction or elimination of the palpable jet is of course important provided it is observed at an adequate blood pressure. Changes in murmurs are influenced by so many factors, including pericardial and pleural effusions in the early postoperative period, that they are often unreliable. The V wave in the pulmonary capillary or left auricular tracing has not correlated at all well, in our experience, with the degree of mitral insufficiency. We are currently investigating differential dye dilution curves during left and right heart catheterization and believe that these may be useful in quantitating regurgitation when correlated with cardiac output studies.

Our point of view concerning the surgical therapy of mitral insufficiency at this time thus differs from that expressed yesterday by Dr. Harken regarding aortic stenosis. We now feel that the majority of these cases are going to require the use of an open technique for constricting the mitral annulus and reducing the herniation of the leaflets.

DR. LEWIS H. BOSHER, Richmond, Va.—I should like to discuss Dr. Glover's paper. Certainly we owe a great deal to Dr. Glover and Dr. Davila, who have developed this procedure for mitral insufficiency. We have operated upon 5 patients with free mitral insufficiency by this technique. These were all advanced cases with severe cardiomegaly. There have been two deaths among the 5, both perhaps preventable deaths with further experience on our part. Certainly the placement of this purse-string suture is difficult, and I think we must all appreciate that a good deal of work in the dog laboratory and also in the post-mortem room is necessary before the procedure is used in the operating room.

In one of our patients, we encountered a pericardial symphysis which made the landmarks difficult to recognize. In this patient, as proved at post-mortem examination six days later, we found that the suture had been passed beneath one of the tricuspid leaflets through the tricuspid orifice and, as it had been tightened, apparently it had come into contact with the ventricular myocardium with subsequent cutting through of the suture as determined at post-mortem. We feel that even if the suture is placed accurately there may be a tendency for it to cut through.

(slide) I think this perhaps illustrates that point. This is a left atrial tracing made at the time of operation before the purse-string suture is tightened. You will notice the high V wave and the high atrial pressures. After the suture is tightened we have a very respectable left atrial tracing. However, on the fourth postoperative day this patient's systolic murmur returned and we felt that perhaps the suture had cut through. Four weeks later we did another left atrial puncture and it is evident that considerable mitral insufficiency has returned. It may be that in this patient there was considerable bunching of the pericardial tube which was used, and this may have accounted for that.

*Ellis, L. B., and Harken, D. E.: Factors Influencing the Late Results of Mitral Valvuloplasty for Mitral Stenosis, Ann. Int. Med. 43: 133-142, 1955.

We have one patient in whom there is certainly a very creditable result. (slide) Here are the preoperative left atrial tracings; here are the tracings three months postoperatively. The mean pressure has dropped from 28 to 7 and the left ventricular pressure has dropped from 15 to 5. This patient has no systolic murmur, the precordium is very quiet, the patient is markedly improved clinically, and we are very happy with the result.

I think in some cases, if considerable tightening of the purse string is necessary in order to eliminate the insufficiency, it may be well to use another technique to supplement this procedure; it may not in itself be entirely sufficient in all cases.

DR. JULIO C. DAVILA, Philadelphia, Pa.—I should simply like to make one comment, perhaps repeating what Dr. Bosher has said; that is, to urge anyone who wishes to attempt this procedure to spend a lot more time than he thinks is necessary in the laboratory prior to attempting it in the patient. I think anyone who does this will find considerable difficulty in learning the technique at first, but as he acquires, shall we say, a spatial concept of the pertinent cardiac anatomy, he will find that the technique is quite easy, with proper orientation.

I should like to make two comments about the point which Dr. Black made, which was a very good one; how to judge the degree of correction of regurgitation. We are not yet at the point where we can measure regurgitation with any degree of accuracy, although some efforts are being made to do so by indicator techniques and these show considerable promise. The jet, per se, is not a satisfactory indicator of how much regurgitation has been modified because the jet will change with changes in systemic pressure. The best evidence of the degree of correction obtained that we have found has been the measurement of left atrial, left ventricular, and aortic pressures, so that we can gauge the degrees of change in the left atrial pressure correlated with the other pressures mentioned.

I would like to emphasize again the technical point that this procedure requires extensive study of the pertinent anatomy. It should be done with precision and with absolute understanding of the anatomy of the heart.

DR. EARLE B. KAY, Cleveland, Ohio.—I should like to discuss Dr. Nichols' very interesting paper. I believe we were one of the first to stress the significance of the dilated mitral annulus and the separated posteromedial commissure in the physiopathology of mitral regurgitation. Over two years ago we developed a technique demonstrating various ways of reducing the size of the annulus at the posteromedial commissure in order to obliterate the regurgitant jet of blood. The particular variation subsequently demonstrated by Dr. Nichols in closing, as illustrative of our technique, was discarded by us for the obvious reasons mentioned. Since the follow-up in their series has been for the most part only several months, I thought it might be of interest to the group to relate the follow-up of 8 patients who were operated upon two years ago employing this principle. The results are about what one would expect. Those patients who had primarily "pure" mitral regurgitation, in whom the valve cusps were fairly pliable so that the commissure could be narrowed, all have done well. Those in whom there was calcification and rigidity of the cusps died, and where the posteromedial commissure could either not be approximated or brought together only under tension, those patients have done poorly and I believe have all died.

In any event, the operation is difficult by the closed technique and not as precise as might be desired. We are hoping that a better result might be obtained with the open direct vision technique employing a pump-oxygenator and following the same basic principles.

DR. RICHARD H. MEADE, Grand Rapids, Mich.—When I was in Dr. Crafoord's Clinic in Stockholm, Dr. Crafoord was away and I saw Dr. Senning, who said, "I am going to try an operation tomorrow on a patient with mitral regurgitation. I am planning to do

a circumferential suture of the annulus." I said, "That is very interesting; I just came from a meeting of the American Association for Thoracic Surgery, where the operation was discussed by Dr. Glover." I wonder if Dr. Senning or Dr. Crafoord have done any more.

DR. H. HANSON, Stockholm, Sweden.—This discussion was not returned. EDITOR.

DR. CHARLES P. BAILEY, Philadelphia, Pa.—First, I want to point out that polar cross-fusion is entirely original with Dr. Nichols—I have had no part in its development except that of offering encouragement. I have had some little part in the handling of some of the cases reported by Dr. Nichols, and I want to emphasize what Dr. Glover has said about the desperate condition preoperatively in most of these individuals. One of the patients in this series was in a hospital under our care for nine months before she could be brought into condition for surgery. Dr. Nichols refuses to talk about the improvement as observed clinically, and yet our cardiologists uniformly are extremely enthusiastic and think the procedure is very good. I am tremendously impressed by the consistently great reduction in the regurgitant jet as recognized by intracardiac palpation at the time of operation. The improvement achieved at surgery transcends anything I have ever been able to accomplish with any other procedure for mitral regurgitation. Of course we must wait to find out what the long-term results will be. I do hope that either Dr. Davila's or Dr. Nichols' procedure will prove to be the final answer. If neither does, we will just have to keep on trying.

I have, as Dr. Glover and Dr. Davila well know, "viewed with alarm" the procedure which they have just reported because I recommended and carried out a somewhat similar operation three and one-half years ago in attempting to correct aortic insufficiency. Those of you who have inserted a Hufnagel valve know how difficult it is to put a circumferential suture accurately about the plastic ring that holds the valve against the aortic wall. This is done under direct vision and with the help of the flanges which are built into the ring. These mitral valve rings are about twice the diameter, are nearly the same thickness, and have no flanges. By a closed, digitally guided technique, it seems inconceivable that a constricting band can be placed accurately about the entire circumference of the mitral (or aortic) ring. Yet, if it slips off any portion of the annulus, it will lose all its constricting effect and, in some instances, will cut through into the lumen. I think my long-term failures with circumferential constriction for aortic insufficiency may be paralleled by similar failures with mitral constriction. Almost all those patients are now dead. The band seemed to slip off the annulus after weeks or months or years. Sometimes it cut through into the aortic or ventricular lumen; other times the regurgitation came back. Nevertheless, if only because they directed our attention toward the importance of the annulus in the correction of mitral insufficiency, we owe Drs. Davila and Glover a tremendous debt.

DR. ROBERT P. GLOVER (Closing).—I should like to thank all the discussants and say a few words in closing. I want to emphasize as strongly as possible the vicious cycle through which mitral insufficiency passes over the years. Any procedure that does not pretty well abolish the existing mitral insufficiency, or if for some reason an appreciable degree of insufficiency remains uncorrected, dilatation of the ring as the years go on will progressively increase. I believe that it is essential to permanently fix the *entire* ring by total constriction; otherwise annular dilatation will continue and the vicious cycle will proceed unabated.

Dr. Black spoke of the aneurysmal dilatation of the left atrium. These atria get as large as the largest of Florida grapefruit and we have been sorely tempted on a number of occasions, after tying the constriction purse-string suture and observing the tense and distended atrial wall to relax and even become wrinkled, to clamp off and resect much of the now redundant atrium. We have not done this as yet, however, so that no confusion would exist as to the proper postoperative evaluation of the purse-string method per se,

but I think it entirely conceivable that some of these tremendous atria can be amputated and refashioned to a reasonable size. These atria no longer beat, they are incapable of contraction, so they might well be made as small as possible.

With regard to Dr. Bosher's statement regarding possible suture cut through into the atrium, such has not occurred when the suture was properly placed. I might state that in some tens of dogs followed well over a year, and we still have twenty to be followed for the next four or five years, there has been no cut through. In our oldest postoperative patient, one on whom an autopsy was performed at eight months, the purse string was intact, well fixed by fibrosis, and no evidence of cut through was present.

The application of circumferential suture to aortic insufficiency is, in my opinion, questionable. The anatomy, pathology, and the actual tissues themselves present in aortic valve disease would not seem to be suitable for a circumferential constriction of this type. The wall of the aortic ring, thin and bare, will not tolerate constant constriction and I believe cut through would be inevitable; but the tissues concerned in the mitral annulus, being muscular as they are, will tolerate constrictive pressures and actually incorporate the purse string within a fibrous capsule. Thus, the least of our problems is concerned with cut through; our greatest problem is the status of the patient's valve itself. Everyone in this audience who has digitally explored a heart valve knows that one cannot predict, prior to such exploration, the actual tissue status of a valve nor what can be accomplished surgically. One can only accomplish what the valve in its diseased state will allow. In 2 of the cases just reported there was actually no remaining functionable valve tissue. The leaflets were contracted right back into the myocardium so that the chordae tendineae actually emerged from a contracted and fixed ridge of scar at the annulus. Despite almost complete constriction of the purse string in these instances nothing but a reduction in size of the mitral orifice could be effected for there were no leaflets to coapt. This situation cannot be predicted clinically and remains for exploration to reveal.

I agree that perhaps total mitral annular purse string is not the final answer, but it is the only procedure of the many tested in our laboratory that routinely will correct regurgitation and it has done so to date, so far as the valves have allowed, in the clinical cases reported.

DR. HENRY T. NICHOLS (Closing).—I should like to thank all the discussants and, if I may, I should like to show a slide.

(slide) In Dr. Kay's discussion, he spoke of having done this same procedure two years before. I think there is a little variation and I have taken the liberty of making this slide from his article. It shows that he has attacked the same portions of the mitral annulus and I certainly give him the credit he deserves. However, I would like to point out that he has used buttons on the outside of the heart in this one of his procedures and that there is considerable soft tissue interposed between the two points of counterpressure, a button on one side and one on the other. I have doubt as to whether the two areas of the annulus will grow together. It might be that this is not important; I do not know. Also, we have worried about the possibility of leaving bare sutures inside the atrium in fear of developing infection.

(slide) Although perhaps this slide is erroneous, I thought he was not pulling the annulus together because of this diagram. I realize that a diagram can be misleading, but it is Kay's diagram and I did get the impression that he was merely distorting the annulus, as shown in these diagrams, before and after.

So far as the history of the attack upon the mitral annulus is concerned, I would like to say that Bernheim, back in 1909, was the first man to attack the mitral annulus, so far as I know. He did this in an attempt to produce mitral stenosis in dogs. He did plicate the annulus, and passed two parallel sutures, one starting from the back of the

heart, piercing the annulus in two places and then emerging from the front of the heart. The second suture was placed parallel to the first. He tied the two free ends on the back together and followed this by tying down the ends in front. Thus he pulled one part of the annulus against the other. In the same article, he described a circumferential suture that he applied. He called it a circular ligature around the mitral annulus, and he passed the needle underneath the coronaries to the left ventricle. I feel that if this is not the same as the Davila technique, it probably is very similar. However, I think Dr. Glover and I, and all of us, can take some comfort in the fact that Bernheim was not successful in producing mitral stenosis.

PATENT DUCTUS ARTERIOSUS IN INFANCY

S. Richard Bauersfeld, M.D.* (by invitation), Paul C. Adkins, M.D.** (by invitation), and Edward M. Kent, M.D.***
Pittsburgh, Pa.

In the usual case of patent ductus arteriosus, there is little if any difficulty during infancy. If the diagnosis is made in early life, operation may be safely deferred until the age of 3 years or more. It is an established fact, however, that a large patent ductus arteriosus may produce severe strain on the circulation, which may ultimately lead to cardiac failure and death during infancy.[6, 7, 10] Consequently, early recognition of this condition is necessary in those infants who are showing evidence of cardiac embarrassment. The purpose of this paper is to discuss those cases of patent ductus arteriosus in which surgical intervention was felt to be indicted before the age of 24 months.

The time of normal anatomic closure of the ductus arteriosus has been discussed in many reviews. Although anatomic patency may persist up to six weeks after birth, most writers feel that functional closure occurs within the first two weeks of life. The ductus which has not closed within the first two months of life will probably remain patent, and the possibility of delayed closure during childhood is remote. The continuous murmur, characteristic of a patent ductus arteriosus, is due to the flow of blood through the ductus during systole and diastole. Since, at birth, there is little difference in pressure between the systemic and pulmonary circulation, there is little flow from the aorta to the pulmonary artery. Consequently, only a systolic murmur is present. With development of the infant, the pressure rises in the systemic circulation and falls in the pulmonary circulation. Eventually, there is a considerable difference in pressure between the aorta and pulmonary artery, resulting in flow of blood through the ductus from the aorta to pulmonary artery during both systole and diastole. At this time, a continuous murmur will be present. Characteristically, these pressure changes occur during the first six months of life, so that prior to the age of 6 months, a systolic murmur only may be present. In some instances, the systolic murmur may not appear until later and, occasionally, may never appear when there is marked pulmonary hypertension.

From the Department of Cardiology, Children's Hospital of Pittsburgh and the Division of Thoracic Surgery, University of Pittsburgh, School of Medicine, Pittsburgh, Pa.

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*Associate Professor of Pediatrics, University of Pittsburgh, School of Medicine and Director of Cardiac Clinic, Children's Hospital of Pittsburgh.

**Instructor in Surgery, George Washington University School of Medicine, Washington, D. C.

***Associate Professor of Surgery and Chief of Thoracic Surgical Section, University of Pittsburgh, School of Medicine.

The findings in a typical case of patent ductus are well known. The most outstanding diagnostic finding is the presence of a continuous murmur at the upper left sternal border. Other salient features are: accentuation of the pulmonary second sound, absence of cyanosis, a wide systemic pulse pressure and fluoroscopic or roentgenographic evidence of increased pulmonary blood flow. In addition, there may be varying degrees of enlargement of the left atrium and left ventricle. Under certain conditions, as previously noted, patency of the ductus arteriosus does not produce a continuous murmur. These patients with only a systolic murmur have been given the term "atypical patent ductus." The term "atypical" is not accurate, however, since the absence of a continuous murmur may be typical for the specific situation, as in young infants. It is not within the scope of this paper to discuss the other causes of a patent ductus arteriosus without a continuous murmur.

Over the past three years, a diagnosis of patent ductus arteriosus was made and confirmed by operation or autopsy on 74 children of all ages at the Children's Hospital of Pittsburgh (Table I). This series does not include any case in which a diagnosis has been made but not yet confirmed by direct visualization. No case was included where a patent ductus arteriosus was present as a compensatory lesion, as in cyantoic heart disease. There are, however, in this series, those cases with an additional anomaly of the heart or great vessels, but in which the patent ductus arteriosus was not considered a compensatory lesion. There were 55 females and 19 males in this series. This corresponds with other reported series, in that there is a preponderance of females. Of the entire group of 74 cases, 22 (30 per cent) were under the age of 2 years. This paper is primarily an analysis of these 22 infants, since it was in this group that the diagnosis was least frequently obvious and, conversely, the same group which presented most often with symptoms which could be attributed to the patent ductus arteriosus. Of the 74 cases of all ages, there were 16 (22 per cent) in whom a systolic murmur only was heard prior to operation. As may be seen from Table I, 12 of the 16 patients in whom a continuous murmur was absent were under the age of 2 years.

TABLE I. AGE, SEX, AND TYPE OF MURMUR IN 74 CASES OF PATENT DUCTUS ARTERIOSUS

AGE GROUP	NUMBER OF CASES	SEX		MURMUR	
		MALE	FEMALE	SYSTOLIC	CONTINUOUS
Under 24 months	22	9	13	13*	9
Over 24 months	52	10	42	4	48
Total	74	19	55	17*	57

*One had systolic and diastolic murmer.

Analysis of the symptoms and physical findings in the children under the age of 24 months (see Table III) shows that nearly all of these children had a history of poor weight gain since birth. Also, many had dyspnea or frequent respiratory infections and 5 gave a history of intermittent minimal cyanosis, usually accompanying respiratory infections. Based on the Boston Children's Medical Center Anthropometric Charts, all but one of these infants were in the third percentile or less for their age and weight. Their birth weights in general were slightly below average and only 4 were under five pounds at birth.

TABLE II. WEIGHT CHANGES IN 22 CASES OF PATENT DUCTUS ARTERIOSUS UNDER 24 MONTHS OF AGE

	PREOPERATIVE				POSTOPERATIVE		
CASE	AGE (MONTHS)	BIRTH WEIGHT (POUNDS)	WEIGHT AT OPERATION (POUNDS)	PERCENTILE	LENGTH OF FOLLOW-UP (MONTHS)	PRESENT WEIGHT (POUNDS)	PERCENTILE*
1. S. M.	5½	6	9½	<3	15	18	<3
2. N. A.	7	6¾	11½	<3	4	18	10th
3. D. D.	10	6¼	11	<3	7	18	<3
4. T. O.	4½	7¼	9¾	<3	26	25	3rd
5. J. N.	16	4¼	10½	<3	12	28	10th
6. L. W.	8	5½	10	<3	23	22	<3rd
7. L. H.	23	5	18	<3	14	25	<3rd
8. R. M.	14	3¾	18	3rd	16	23½	<3
9. M. B.	8	4¼	9	<3	Coaretation and PDA; Died 3 mos. postoperative		
10. G. G.	4	8¾	12	<3	PDA and IASD; Died 3 mos. postoperative		
11. M. S.	2	6	6½	<3	7	10½	<3
12. P. K.	6	8	11	<3	No operation		
13. J. S.	20	9½	16½	<3	27	39	>75<90
14. S. B.	6	5	10¼	<3	3	16	10th
15. A. F.	8	5¾	15	3rd	18	22	3rd
16. C. H.	21	3½	16	<3	11	20½	<3
17. R. C.	7	5¼	9	<3	10	21½	>10<25
18. N. M.	12	8½	18	25	1		
19. K. B.	3	6¾	6½	<3	No operation		
20. M. W.	2	6	7½	<3	1	9½	<3
21. F. Y.	6	4¾	8¾	<3	1		
22. D. N.	5	5¾	10½	<3	1		

*Based on The Boston Children's Medical Center Anthropometric Charts.

Table III lists the physical findings in this group of 22 infants. In 9 (41 per cent) of these infants, a continuous murmur was heard. Of the remaining 13 infants, 12 had only a systolic murmur located along the left sternal border. In one infant, the murmur was systolic and diastolic but not continuous. In most of this group, the thrill and murmur were of maximal intensity in the pulmonic area. However, in 3 infants (Cases 2, 9, 10) with proved patent ductus arteriosus, the thrill and murmur were maximal along the lower left sternal border. All showed roentgenographic evidence of cardiac enlargement and increased pulmonary vascular markings. In those infants where blood pressure was obtained by the flush technique, the systolic pressure only is listed.

The electrocardiogram showed considerable variation (Table IV). In 16 cases the axis was normal, 11 of these had no hypertrophy from the precordial leads; in 3 there was left ventricular hypertrophy and in 2, right ventricular hypertrophy. Right axis deviation plus right ventricular hypertrophy was present in 5 infants. In one infant, there was left axis deviation and no evidence of ventricular hypertrophy from the precordial leads. Four of the 7 cases with right ventricular hypertrophy had additional cardiac anomalies; one with coaretation, 2 with interventricular septal defects and one with reversed flow through the ductus. There was no evidence of additional cardiac anomalies in the remaining 3 cases. One of this group died and, at autopsy, the only abnormality found was a large patent ductus arteriosus.

TABLE III. SIGNS AND SYMPTOMS IN 22 PATIENTS UNDER 24 MONTHS OF AGE WITH PATENT DUCTUS ARTERIOSUS

	HISTORY				PHYSICAL FINDINGS			
					PULMONARY SECOND SOUND	MURMUR		BLOOD PRESSURE
CASE	POOR WEIGHT GAIN	RESPIRATORY DIFFICULTY	INTERMITTENT CYANOSIS	THRILL		CONTINUOUS	SYSTOLIC	
1. S. M.	+	+	+	0	Inc	—	2 and 4 LICS	—
2. N. A.	+	+	0	+	Inc	—	4th LICS	120/80
3. D. D.	+	0	0	+	Inc	—	3 and 4 LICS	—
4. T. O.	+	0	0	0	Inc	2 and 3 LICS	—	100
5. J. N.	+	0	+	+	Inc	—	2nd LICS	86
6. L. W.	+	+	0	0	Inc	—	2 and 3 LICS	94
7. L. H.	+	0	0	+	Inc	—	LSB	130/80
8. R. M.	+	0	+	0	Inc	—	LSB	70
9. M. B.	+	0	0	0	Inc	—	4th LICS	Arm 115 Leg 50
10. G. G.	+	+	+	0	Inc	—	4 and 5 LICS	115/65
11. M. S.	+	+	0	0	Inc		LSB	—
12. P. K.	+	+	+	+	Inc	3rd LICS	—	75
13. J. S.	+	0	0	+	Inc	2nd LICS	—	120/60
14. S. B.	+	0	0	0	Inc	2nd LICS	—	—
15. A. F.	0	0	0	+	Inc	2 and 3 LICS	—	100/50
16. C. H.	+	+	0	0	Inc	2 and 3 LICS	—	—
17. R. C.	+	+	0	+	Inc	2 and 3 LICS	—	100
18. N. M.	0	0	0	+	Inc	2 and 3 LICS	—	—
19. K. B.	+	+	0	0	Inc	—	LSB	
20. M. W.*	+	0	0	+	—			120/60
21. F. Y.	+	0	0	+	Inc	2nd LICS	—	140
22. D. N.	+	+	0	0	—	—	3 and 4 LICS	120
Total	20	10	5	11	20	9	12*	—

*Systolic and diastolic but not continuous at 4 LICS.

Key: Inc—increased.
LICS—left intercostal space.
LSB—left sternal border.

TABLE IV. SPECIAL STUDIES IN 22 CASES OF PATENT DUCTUS ARTERIOSUS UNDER 24 MONTHS OF AGE

	X-RAY AND FLUOROSCOPY						ELECTROCARDIOGRAM			
CASE	C-T RATIO	LV	LA	RV	RA	PULMONARY VASCULARITY	AXIS	PRECORDIAL LEADS	AORTOGRAM	FINAL DIAGNOSIS
1. S. M.	.60	+	0	0	0	Inc	BAL	LVH	PDA	PDA
2. N. A.	.63	+	±	0	0	Inc	BAL	—	PDA	PDA
3. D. D.	.58	++	+	0	0	Inc	BAL	LVH	PDA	PDA
4. T. O.	.55	++	+	0	0	Inc	BAL	—	PDA	PDA
5. J. N.	.62	±	0	++	±	Inc	RAD	RVH	PDA	PDA
6. L. W.	.62	++	+++	0	+	Inc	BAL	RVH	Unsatis.	PDA and IVSD
7. L. H.	.58	+	0	0	0	Inc	BAL	—	—	PDA
8. R. M.	.56	+	0	+	±	Inc	RAD	RVH	—	PDA
9. M. B.	.58	+++	0	+	0	Inc	RAD	RVH	PDA and Coarc.	PDA and Coarc.
10. G. G.	.63	+	0	0	0	Inc	LAD	—	PDA	PDA and IASD
11. M. S.	.60	±	0	+++	0	Inc	RAD	RVH-LVH	PDA	PDA and IVSD
12. P. K.		+	0	+	+	Inc	RAD	RVH	—	PDA
13. J. S.	.58	±	0	0	0	Inc	BAL	—	—	PDA
14. S. B.	.60	±	0	+	0	Inc	BAL	RVH	—	PDA
15. A. F.	.60	++	++	0	0	Inc	BAL	—	—	PDA
16. C. H.	.53	+	±	++	++	Inc	BAL	—	—	PDA
17. R. C.	.6–	++	++	0	0	Inc	BAL	—	—	PDA
18. N. M.	.54	++	++	0	0	Inc	BAL	—	—	PDA
19. K. B.	.63	++	++	±	++	Inc	BAL	—	—	PDA
20. M. W.	.50	+	+	+	0	Sl. Inc	BAL	LVH	PDA	PDA
21. F. Y.		0	±	0	0	Sl. Inc	BAL	—	—	PDA
22. D. N.	.50	+	+	0	0	Inc	BAL	—	PDA	PDA

Key: C-T Ratio—Cardio-thoracic ratio.
LVH—Left ventricular hypertrophy.
RVH—Right ventricular hypertrophy.
BAL—Balance.
LAD—Left axis deviation.
RAD—Right axis deviation.

On the basis of a continuous murmur in the pulmonic area and other substantiating evidence by physical examination and fluoroscopy of the heart and lungs, it was possible to make a diagnosis of patent ductus arteriosus in 9 of these infants when first seen. In the remaining 13 infants, the physical findings were suggestive of a patent ductus arteriosus; in these cases, however, the absence of a continuous murmur made it impossible to differentiate this from other

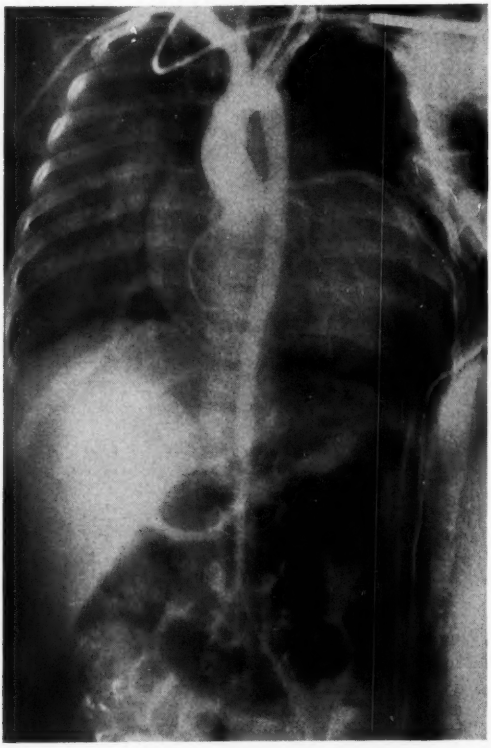

Fig. 14.—Normal aortogram.

lesions causing a left-to-right shunt. The lesion we considered most difficult to differentiate from a patent ductus arteriosus in these infants was a high ventricular septal defect. Both a patent ductus arteriosus and a high ventricular septal defect may present with a similar history of poor weight gain, frequent respiratory difficulty, and a heart murmur heard during early life. Both conditions may present a systolic thrill and murmur heard along the left sternal border and a loud pulmonary second sound. The location of the thrill and murmur may not be helpful in differentiating between a patent ductus and a ventricular septal defect in these infants, since in several of our cases with proved patent ductus arteriosus, the thrill and systolic murmur were heard at the lower left

sternal border (Cases 2, 9, 10). Both conditions may exhibit increase in the over-all heart size and fluoroscopic evidence of increased pulmonary blood flow.

Consequently, in those infants seen with the above findings, who showed evidence of poor development and cardiac enlargement, it was felt that additional studies were indicated if a patent ductus arteriosus could not be ruled out as the cause of the left-to-right shunt. Since the infants in this group were quite small, the average weight being eleven pounds, retrograde aortography was considered more practical than cardiac catheterization to establish the

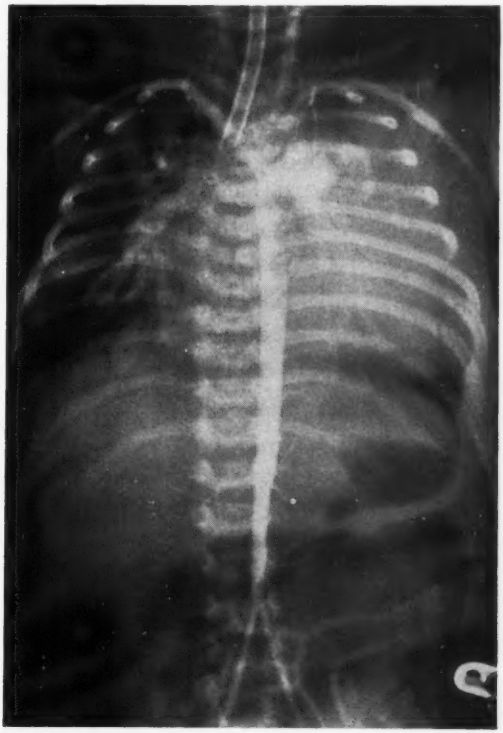

Fig. 1*B*.—Aortogram showing simultaneous filling of the aorta and pulmonary artery.

presence or absence of a patent ductus arteriosus. One infant (Case 8) had findings suggestive of a patent ductus arteriosus with reversal of flow. This infant had rather striking intermittent cyanosis which was more marked in the lower extremities. In this case the diagnosis was confirmed by angiocardiogram. The diagnosis of patent ductus arteriosus was established by retrograde aortogram in 9 infants.

In the majority of cases, retrograde aortography was carried out under general endotracheal anesthesia and a modification of the method described by Keith was used. The carotid sheath was exposed through a 1.5 cm. transverse

incision at the anterior border of the left sternocleidomastoid muscle. The left common carotid artery was freed up and two heavy silk ligatures were passed around it but not tied. Using these ligatures to control bleeding, a 3 mm. longitudinal opening was made in the carotid artery. A No. 240 polyethylene catheter was then passed proximally in the carotid artery for a distance of 2 to 3 cm. The lower ligature was then tied once around the vessel to hold the catheter in place. It was found by trial and error that a better concentration of the contrast medium was obtained if the tip of the catheter lay somewhat above the junction of the aorta and the left common carotid artery. After giving a test dose to determine sensitivity, a rapid injection of 6 to 8 ml. of 35 per cent Diodrast was made into the carotid artery through the cannula, and a single anteroposterior chest x-ray taken at the exact moment of completion of the injection. During injection of the dye, the opposite common carotid artery was compressed by the anesthesiologist. After the film had been taken, the catheter was withdrawn and the opening in the vessel closed with a continuous suture of 6-0 arterial silk. The remainder of the incision required only two or three fine silk sutures for closure. Simultaneous demonstration of the dye in the aorta and pulmonary arteries by x-ray was considered diagnostic of an aorticopulmonary shunt (Fig. 1.).

Using this technique, retrograde aortography was performed on 43 infants during this three-year period to determine the presence or absence of a patent ductus arteriosus. There were two deaths which could be ascribed to aortography, both early in the series, an over-all mortality of 4.6 per cent. It was felt that one of these deaths was definitely associated with an overdose of the contrast medium. The other infant demonstrated an interventricular septal defect, coarctation of the aorta, and subendocardial fibroelastosis at autopsy. If a patent ductus arteriosus was demonstrated by aortography, surgical closure was usually carried out within a week.

In one infant, the aortogram was technically unsatisfactory, but a patent ductus arteriosus was demonstrated at operation. In one case, a coarctation of the aorta and a patent ductus were suspected before aortography and this diagnosis was confirmed. In another, a questionable coarctation was seen on the aortogram, but there was no evidence of narrowing of the aorta at operation.

Of the 22 infants with patent ductus arteriosus, 2 died before any definitive procedure could be carried out. One infant (Case 12) was admitted with pneumonia and found to have the physical findings of a patent ductus arteriosus. Despite intensive efforts, the infection could not be controlled to a point where the patient was considered a suitable candidate for anesthesia and the child died before operation could be carried out. Autopsy revealed only a large patent ductus arteriosus. Another infant (Case 19) was admitted with a severe respiratory infection. Although a continuous murmur was not audible, the physical findings were suggestive of a patent ductus arteriosus. Retrograde aortogram was contemplated, but before the respiratory infection could be controlled, the infant died suddenly. At autopsy, an uncomplicated patent ductus arteriosus was demonstrated.

Operation was carried out on the remaining 20 infants in this series. Those patent ducti which were 10 mm. or more in width were divided between clamps and the ends closed by continuous suture. The patent ducti smaller than 10 mm. in width were closed by the multiple ligature technique.[12] There were no operative deaths. In the infant (Case 8) with reversal of flow through the patent ductus, temporary occlusion of the ductus at operation produced no deleterious effect and division was carried out without difficulty. This patient has continued to progress satisfactorily.

In 3 infants, there was a residual thrill over the base of the heart after occlusion of the patent ductus. One of these infants (Case 10) died three months postoperatively and was found to have a large residual interatrial septal defect. The other 2 (Cases 6 and 11) are living and well and both are considered to have a residual interventricular septal defect. The one infant (Case 9) who had a coaretation of the aorta and an associated patent ductus arteriosus proximal to the coaretation, died three months postoperatively. In this case, resection of the coaretation, as well as division of the patent ductus, had been contemplated prior to operation. However, after divisions of the patent ductus, the infant's condition became so precarious that further surgery was precluded and the chest was closed. Therefore, since only the patent ductus arteriosus was divided in this infant, the case is included in this series.

Postoperative follow-up on the remaining 18 infants, over a period from one to twenty-seven months, reveals that all have done well. Nearly all have gained weight satisfactorily, have been less prone to respiratory infections, and have been less irritable.

SUMMARY

Persistent patency of the ductus arteriosus is most frequently asymptomatic during infancy and early childhood. However, in some infants, a patent ductus arteriosus may cause failure to gain weight, respiratory infections, and cardiac enlargement. Many of these infants with symptoms referable to the patent ductus do not have a continuous murmur. Consequently, the correct diagnosis is not obvious. The physical findings in this group are similar to the findings in infants with left-to-right shunts at other levels. In these infants, when evidence of cardiac enlargement is present, the diagnosis must be established by special studies. During the period that this series was seen, operative procedures for closure of septal defects were not available to us. Consequently, retrograde aortography was considered the simplest method of establishing the presence or absence of a patent ductus arteriosus in small infants. With the successful development of techniques for open cardiotomy, cardiac catheterization is indicated to establish the exact level of the left-to-right shunt.

It is also felt that if an infant has a left-to-right shunt at two levels, such as an interventricular septal defect and a patent ductus arteriosus, these have an additive effect. Consequently, definite benefit may be obtained by closure of the patent ductus arteriosus.

The frequency of patent ductus arteriosus without a continuous murmur is somewhat higher in this group than in other reported series.[8] The majority of cases presenting with systolic murmurs alone were infants. Consequently, it is felt that a diagnosis of patent ductus arteriosus should be considered in noncyanotic infants presenting with a systolic murmur and a history of poor weight gain. If there is evidence of increased pulmonary vascularity and cardiac enlargement, the diagnosis may be established by retrograde aortogram. If a patent ductus arteriosus is demonstrated, surgical closure can be performed an a normal cardiovascular status restored.

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Discussion

DR. FRANCIS DAMMANN, JR., Charlottesville, Va.—I certainly enjoyed this paper, as I have the entire meeting. I am very glad that I was privileged to come.

There are a few points I would like to re-emphasize and add in addition to what Dr. Adkins has said. It is very true that patent ductus in early infancy can cause death. It is true that unless one has a high level of suspicion, in many instances, the diagnosis will not be made or even thought of. Once it has been suspected I agree with the authors that the best and easiest technique for establishing the diagnosis is retrograde aortography. We have injected a 35 per cent solution of Diodrast into the left, not the right brachial artery, feeling that perhaps this keeps it away from the brain and may cut down the mortality. Obviously, this procedure does not rule out an aorticopulmonary shunt which may be missed unless the right brachial artery is used. I think we have established the diagnosis of patent ductus in about one half of the patients who have had aortograms. I would consider this an acceptable percentage because the frequency of cardiac decompensation and death in this group is high.

There is another aspect that I think should be mentioned. We have seen several infants who have come out of cardiac failure and then developed a right-sided shunt. This indicates progressive pulmonary vascular disease. It seems to me that, if this process is to be avoided, early intervention, early interruption of the high pressure in the lungs, is very definitely indicated.

MR. RAYMOND HURT, London, England.—I should like to report 26 cases of patent ductus arteriosus in infancy that have been operated upon by Dr. Frank Gerbode in San Francisco. These infants have all been admitted to hospital suffering from recurrent attacks of heart failure associated with respiratory infection. Almost all these patients had only a systolic murmur over the precordium, together with clinical evidence of heart failure. The diagnosis has been confirmed by retrograde aortograms done through the left brachial artery, by making a small incision under local anesthesia and introducing a needle into the artery under direct vision. Of the total number of infants studied approximately one fourth have had a surgically correctible lesion. All these ducti have been divided and there has been no mortality. The benefit from the closure has been most satisfactory, even in those cases that have had an associated abnormality.

DR. EGBERT FELL, Chicago, Ill.—I wish to re-emphasize the importance of the paper that was so well presented by Dr. Adkins. We have learned the hard way regarding these little infants, and to date we have had 18 infants, one year of age or less, with large open ducti. The first 2 were not recognized and came to post-mortem, 14 are living normally following closure of the ducti, the seventeenth was found to have an aortic septal defect which was successfully closed, and the eighteenth died three hours after surgery in heart failure believed due to overhydration.

We do not believe aortography is indicated, and believe that its use carries an unnecessary risk of major importance. Dr. Benjamin M. Gasul, our pediatric cardiologist, believes and clearly demonstrates that this anomaly can be diagnosed clinically in almost every patient. The infant is sedated so that a careful satisfactory physical examination, electrocardiographic and x-ray studies can be done. The machinery murmur is usually heard and a wide pulse pressure found. The heart is enlarged and pulmonary vascular markings are increased. If we carry out a thorough examination with a cooperative sedated infant, more successful diagnoses and cures of the so-called "malignant ductus" will be reported.

DR. H. HANSON, Stockholm, Sweden.—This discussion was not returned. EDITOR.

DR. F. MASON SONES, JR., Cleveland, Ohio.—Over a six-year period, we have encountered 21 infants younger than one year of age, who proved to have patent ducti and who did not have continuous murmurs. I feel very strongly that the diagnosis in this situation is best made by cardiac catheterization. All these infants were studied by careful auscultation under excellent conditions, and in none of them were we able to recognize a continuous murmur. We were unable to tell the difference between a huge patent ductus and a large interventricular septal defect. In 20 of the 21 infants in whom the diagnosis was proved, it was possible actually to catheterize the ductus. This can be done safely and is not difficult from a technical standpoint. It seems to me that the major factor in favor of retrograde aortography is that it can be accomplished quickly and with little loss of time. Catheterization certainly is more painstaking and does take a great deal more time, but offers a greater margin of safety to the patient than is possible with retrograde aortography.

DR. HERBERT E. WARDEN, Minneapolis, Minn.—I rise to compliment Dr. Bauersfeld and his coauthors for their very fine paper. In addition, I would like to thank them for stressing the importance of the patent ductus arteriosus in the infant age group, and to reaffirm their feeling that the surgical closure of a patent ductus in the presence of an associated interventricular defect, as it often is, nets a marked clinical improvement in the patient. This is often true even if nothing is done for the septal defect.

The patent ductus arteriosus has been the most frequent associated extracardiac congenital anomaly encountered in the group of patients that we have seen with septal defects. To date all but one of these occurred in patients in this young age group. Preferentially, we feel that the ductus should be closed at the same time that corrective surgical repair of the ventricular septal defect is carried out. However, if this is not feasible and

the patient's condition is deteriorating, a remission or at least a retardation or a slowing of the downhill course can be accomplished by the closure of the ductus even in the presence of a persistent ventricular shunt. We have seen this occur in one patient who was moribund. The presence of an interventricular defect was not suspected in him prior to thoracotomy and therefore suitable preparations for intracardiac repair were not available. However, the ductus was transected and the patient made a remarkable recovery following this closure and some months later successfully endured the surgical repair of the interventricular septal defect.

DR. H. BRODIE STEPHENS, San Francisco, Calif.—I enjoyed this presentation and believe it would be advisable, when we discuss this subject of patent ductus arteriosus in infants, that measurements be made of the diameter of the ductus by calipers and, in addition, measurement of the diameter of the aorta and pulmonary artery be carried out at the same time. I think by so doing and by documenting and reporting these measurements, we might obtain additional information on this interesting subject.

DR. S. RICHARD BAUERSFELD (Closing).—I certainly appreciate all the discussion of this paper which Dr. Adkins presented. I should like to make one or two comments about some of the statements that have been made.

First of all, we agree with Dr. Dammann about the importance of being aware of an atypical ductus. The diagnosis becomes easier when one realizes that a ductus can cause great difficulty and he is willing to hunt for this lesion. If one does not take all this trouble, one will not find it. We are indebted to Dr. Dammann for the initial studies of this condition. He reported a series of ducti very similar to our group in *Circulation* in 1952 or 1953. In our own group of patients the majority were less than one year of age, only 5 being over twelve months.

We agree with Dr. Fell that the pulse pressure is very important, although we have seen very large pulse pressures in patients with large ventricular defects. I certainly cannot agree with him about the continuous murmur. Likewise, in a recent report in *Pediatrics* from the Children's Hospital in Philadelphia, a large series of ducti were reported in which continuous murmurs were found in the majority of the patients. It has been our policy to go into the operating room and listen to these patients while they are under anesthesia, yet we have not been able to find continuous murmurs even though we know that a ductus is present. In this regard we agree with Dr. Sones that aortograms are necessary to demonstrate the ductus.

So far as measurements of the ductus are concerned, unfortunately we have not done this. We have approximated measurements. As a rule the ducti are as large as the aorta and in several cases the ductus was actually larger than the aorta. The statement about the risk of aortograms—this is true. It does carry a risk and in our own series this comes to some 5 per cent. On the other hand, the risk of missing the diagnosis of a ductus, and having the patient die, is even greater. Therefore, balancing these things, we certainly feel that aortography should be done. One thing that has struck us is that associated anomalies in these children are usually higher than are reported in other series of ducti. In our over-all group of some 74 patients, the incidence of additional defects amounted to 10 per cent. In this particular group it is almost 20 per cent.

At one time we thought the finding of right ventricular hypertrophy would help differentiate a patient with a patent ductus from one with an interventricular septal defect. However, as pointed out, 3 of our patients had right ventricular hypertrophy and nothing but a large ductus. At the present time, I do not believe that I can separate a patient with a ventricular defect from one with a ductus without going ahead with additional studies such as aortography.

Letters to the Editor

Recently, the Editor received a letter from Dr. Seymour Schotz of The Presbyterian Hospital in Philadelphia objecting to the discussion made by Dr. Jerome R. Head, of Chicago, of the papers dealing with anesthesia that were published in the October issue of the JOURNAL. Dr. Head's remarks were as follows:

"The presentation of these interesting and excellent papers before this Association seems to me important and appropriate. However much the anesthesiologists have tried to argue us out of the contention, the responsibility for the anesthesia lies with the surgeon, and for the surgeon to pass it entirely to the anesthesiologist is against Hippocratic principles and against the principles of modern law. The findings presented in these papers also seem encouraging. It means that what we had almost come to accept as a fated and unavoidable accident is becoming an understandable phenomenon and a preventable phenomenon. Among all primitive peoples, when anything is not understood and is potentially disastrous, it is attributed to evil spirits, and I would confess that my attitude toward cardiac arrest has been heretofore largely on that basis. One of the evil spirits has been the anesthesiologist, but more specifically and less facetiously, for a long time I placed my faith in ether. As a young man I went into the operating room and was given a can of ether and a mask and told to put people to sleep. They all lived, which was very surprising to me, and I attributed it entirely to the fact that you could not kill people with ether. But I found out, over a period of years, that I had had three cardiac arrests under pure ether anesthesia, all of them during induction. I am sure now that this was because of inadequate relaxation and of multiple attempts to insert an intratracheal tube. I was finally convinced that a long difficult induction with multiple attempts to insert a tube was bad. I should have said also that one of my evil spirits was curare; I turned to Pentothal and nitrous oxide and to controlled respiration, and I put my faith in that, as one would put his faith in a god or a dance or whatever you have. I got along very well with that until I realized that controlled respiration was of two kinds, either manual or mechanical. Recently I had a case of cardiac arrest due, I feel sure, to the fact that the anesthesiologist became so interested in watching the resident put a catheter into a vein in the leg that he forgot to pump the bag. That is a human error and, as a result of it, I have gone to mechanical respiration and to hyperventilation. Hyperventilation may have certain drawbacks but they are much less than those of underventilation. Therefore, at the present time, as soon as I make the incision I begin to say—'The blood is black, come on, get going, the blood is black.' I do that at intervals all through the operation and so far it has worked. I have not had a cardiac arrest for a year.

"These papers are very interesting and very important but they do not show what happens during cardiac arrest. I feel that every anesthetic agent at the present time should be considered experimental, that any anesthesiologist who is not checking himself and being checked by blood oxygen readings, by CO_2 analyses, and by the running electrocardiogram, is not being a good anesthesiologist. The only way in which we will learn exactly what happens, exactly what causes cardiac arrest, is to have such data on a group of cases that have suffered cardiac arrest."

The letter from Dr. Schotz is as follows:

To the Editor of THE JOURNAL OF THORACIC SURGERY:

It is difficult to read Dr. Head's offensive remarks which appeared on page 476 of the October, 1956, issue of THE JOURNAL OF THORACIC SURGERY without saying to one's self "He's only joking," and letting it go at that. But since this form of wit (which used to be so prevalent in the past and which fortunately seems to be disappearing from the average surgeon's armamentarium) ceases to be funny in this era wherein those of us who are keenly interested in advancing the caliber of anesthesia practice are laboring to attract brighter minds to the specialty, it deserves some comment.

Dr. Head states that for the surgeon to relinquish the anesthetic care to another person would be violating his Hippocratic oath. May I point out that for something close to 80 years, following the discovery of ether, surgeons in general did a fairly thorough job of relinquishing this responsibility by paying almost no attention to the problems of disturbance of physiologic processes which occur under anesthesia. These studies awaited the appearance on the scene of an inquisitive group of physicians who called themselves anesthestists. Surgeons in fact went even one step further and allowed the important task of rendering patients unconscious to fall into the hands of ill-trained nurses and inexperienced doctors. I am told that one famous surgeon, perhaps even as famous as Dr. Head, used his chauffeur for his anesthestist. I would say that it would take something like a perverted sense of values to look upon such practices as carrying out one's Hippocratic responsibilities. On the contrary there seems to be a strong feeling going about that the successful carrying out of many surgical manipulations calls for the most intimate collaboration of surgeon and anesthetist, each performing his task independently and in concert, that it is a physical impossibility for the surgeon to perform both tasks well unless he has a very marked divergent squint and could move from the operating position to the head of the table with the speed of an electronic switching device. With all due respect to Dr. Head's fame as a surgeon, I doubt he is capable of doing that. In no branch of surgery has this intimate collaboration between the well-trained physicians in surgery and anesthsia proved itself more essential than in the field of thoracic surgery, where both are literally working in the same area. Dr. Head's suggestion that cardiac arrest is best prevented by a constant reference to blood being dark is hardly the answer to any question much less the serious question of the prevention of this catastrophe. It strikes me that it has been just the sort of practice which Dr. Head recommends which has been as responsible as anything else in failing to bring to light some of the important aspects of the alteration in human physiology under the stresses of anesthesia and surgery. We need clear and scientific minds for this problem, not minds warped by seeing this or that as an evil spirit.

Dr. Head's final remark was so important to my way of thinking that I wish he had said just this and nothing else. He refers to the need for special equipment for determining the oxygen saturation of the arterial blood, for monitoring CO_2 in inhaled mixtures and for a continuous perusal of the electrocardiogram, that all this should be the routine in good anesthesia practice. It is an interesting fact that many of these devices are on the market and could be used a good deal more routinely, at least in major intrathoracic operations. May I point out that most of these instruments are extremely expensive and can hardly be paid for in so much operating room feet. There is a crying need for reappraisal of the funds allocated for all the fancy trimmings which seem to be a part of the modern hospital with perhaps a greater allocation of funds for the purchase of these instruments which would tell us more about our patients. Now there is an evil spirit for Dr. Head to attack.

Very truly yours,

SEYMOUR SCHOTZ, M.D.
Chief, Department of Anesthesiology
The Presbyterian Hospital in Philadelphia

The Editor sent a copy of Dr. Schotz's letter to Dr. Head informing him that if he wished he could publish a reply. Dr. Head's reply is as follows:

To the Editor of THE JOURNAL OF THORACIC SURGERY:

I quite agree with you that the discussion of our papers should be open to discussion, and I would be very glad to have you print Dr. Schotz's letter concerning my discussion. I am very sorry that he took offense where no offense was intended. I have reread my discussion and fail to see that it could reasonably give offense.

I agree with Dr. Schotz that the problem of cardiac arrest under anesthesia is a tremendously serious matter. I disagree with him only in his statement that the specialty of anesthesiology is in need of brighter minds. My experience with anesthesiologists has been very good and I am convinced that they have contributed importantly to the recent great advances in surgery. However, they are only human, and when I see that the blood is dark, as I too frequently do even without turning my head toward the head of the table, I feel that I am justified in being alarmed and calling attention to it; and inasmuch as I am convinced that anoxia and hypercapnia are extremely dangerous, and hyperventilation is infinitely less dangerous, I feel justified in preferring to have my patients hyperventilated rather than hypoventilated. Furthermore, I do not see how any surgeon can be other than deeply concerned about the fact that cardiac arrest during anesthesia has become one of the chief causes of death in surgery. The patients come to the surgeon for treatment of their diseases; reasonably and legally he is the only one basically responsible to see that they come through the ordeal alive and well. I believe that it is my attitude on this point which was chiefly offensive to Dr. Schotz.

If you decide to print Dr. Schotz's letter, I would appreciate it very much if you would reprint my discussion and print this letter with it.

Very sincerely yours,

JEROME R. HEAD, M.D.
55 East Washington Street
Chicago 2, Illinois

American Association for Thoracic Surgery

CONSTITUTION

(As Amended to May 8, 1956)

Article I. Name

SECTION 1.—This Association shall be known as the American Association for Thoracic Surgery.

Article II. Object

SECTION 1.—The object of the Association shall be to encourage and stimulate investigation and study that will increase the knowledge of intrathoracic physiology, pathology, and therapy, to correlate such knowledge and disseminate it.

SECTION 2.—To attain this object, the Association shall hold at least one scientific meeting every year in which free discussion shall be featured; shall conduct a Journal for the publication of the papers presented at this meeting, and other acceptable articles; and shall undertake such other activities as the Council or the Association as a whole may decide.

Article III. Membership

SECTION 1.—There shall be four classes of members: Active, Associate, Senior, and Honorary. Admission to membership in the Association shall be by election. There shall be no numerical limits on any class of membership. Only Active and Senior members shall have the privilege of voting or holding elective office.

SECTION 2.—Election to all classes of membership shall be for life, subject to the provisions of Section 3, following.

SECTION 3.—Membership may be voluntarily terminated at any time by members in good standing. The Council, acting as a Board of Censors, may recommend the expulsion of a member on the grounds of moral or professional delinquency, and submit his name, together with the grounds of complaint, to the Association as a whole at any of the regularly convened meetings, after giving the member so accused ample opportunity to appear in his own behalf.

Article IV. Officers and Government

SECTION 1.—The officers of the Association shall be a President, a Vice-President, a Secretary, a Treasurer, an Editor, and five Councilors. These ten officers and councilors shall be the governing body of the Association, and shall have full power to act on all matters, except as follows:

1. They may not alter the initiation fees or annual dues, nor levy any general assessments against the membership, except that they may, in individual cases, remit annual dues or assessments.

2. They may in no wise change the Constitution or By-Laws.

3. They may neither elect new members nor alter the status of existing members, other than to apply the provisions of Article III, Section 3.

4. They may not deplete the principal of the Endowment Fund.

SECTION 2.—Officers and Councilors shall be elected at the annual meeting of the Association, and shall take office upon conclusion of the meeting. The President and the Vice-President shall be elected for a one-year term of office and neither may be re-elected to succeed himself in the same office. The Secretary, the Treasurer, and the Editor shall be elected for a one-year term of office, and any or all may be re-elected indefinitely. The outgoing President shall automatically become a Councilor for a one-year term of office. The other four Councilors shall be elected, one each year, for a four-year term of office, but no Councilor may be re-elected to succeed himself.

SECTION 3.—Vacancies occurring among the officers and councilors during the year shall be temporarily filled by action of the Council, subject to approval of the Association at the next regularly convened meeting.

ARTICLE V. COMMITTEES

SECTION 1.—At the opening session of the annual meeting there shall be elected, after nomination from the floor of the Association, a Nominating Committee of three. This Committee shall prepare a slate of nominees for officers and councilors and shall present their report at the executive session of the Association.

SECTION 2.—The Council is empowered to appoint a Membership Committee, an Auditing Committee, a Program Committee, a Necrology Committee, and such other committees as may in its opinion be necessary. All such committees shall render their report at the executive session of the Association.

SECTION 3.—The Editor is empowered to appoint an Editorial Board, subject only to the approval of the Council.

SECTION 4.—The Association as a whole may authorize the Council to appoint Scientific or Research Committees for the purpose of investigating thoracic problems and may further authorize the Council to support financially such committees to a limited degree. In appointing such committees, the Council shall be governed by the provisions of the By-Laws.

ARTICLE VI. FINANCES

SECTION 1.—The fiscal year of the Association shall run from the beginning of one annual meeting to the beginning of the next annual meeting. The books of the Association shall be kept and audited on this basis.

SECTION 2.—Members shall contribute to the financial maintenance of the Association through the medium of initiation fees, annual dues, and special assessments. The amount of the annual dues and the initiation fees shall be determined by the By-Laws.

If, at the end of any fiscal year, there be a deficit in the current funds of the Association, the Council may send out notices to that effect and invite Ac-

tive Members to contribute the necessary amount so that no deficit be carried over from one fiscal year to another. The Association may, in any regularly convened meeting, vote a special assessment for any purpose consistent with the objects of the Association (Article II), and such special assessment shall become an obligatory charge against the classes of members affected thereby.

SECTION 3.—To meet the current expenses of the Association, there shall be available all revenue derived from annual dues, special assessments, and income from Endowment Fund, subject to the provisions of Section 4, following. Funds derived from the payment of initiation fees shall not be available for current expenses.

SECTION 4.—All funds derived from the payment of initiation fees shall be placed in a special fund, to be invested and reinvested in legal securities, to be held intact, and to be known as the Endowment Fund. The Council is responsible for the proper management of the Endowment Fund, and may divert any surplus in the current funds of the Association into this fund, but may not withdraw any of the principal of the Endowment Fund except in accordance with the provisions of Section 6, following.

SECTION 5.—The income from the Endowment Fund shall be expended as the Council directs.

SECTION 6.—The principal of the Endowment Fund may be withdrawn, in whole or in part, under the following conditions only: The amount of principal to be withdrawn shall have been approved by the Council; it shall have been approved by a majority of the members present and voting at a regularly convened annual meeting; it shall have been tabled for one year; it shall have been finally passed by a three-fourths vote of the members present and voting at the next regularly convened annual meeting.

SECTION 7.—In the event of the dissolution of the Association, the Endowment Fund shall be distributed among national institutions of the United States and Canada in a proportion equal to the then existing ratio between the numbers of citizens of the two nations who are members of the Association.

ARTICLE VII. MEETINGS

SECTION 1.—The time, place, duration, and procedure of the annual meeting of the Association shall be determined by the Council, and the provisions of the By-Laws.

SECTION 2.—A special meeting of the Association may be called on one month's notice on the written request of fifteen members. The specific purposes of the meeting must be stated in the request and in the official call for the meeting.

SECTION 3.—The annual meeting of the Council shall be held at or near the close of the fiscal year.

ARTICLE VIII. AMENDMENTS

SECTION 1.—This Constitution shall in no wise be changed except by a three-fourths vote of the members present at an annual meeting, and further

provided that the proposed alteration or amendment shall have been moved and seconded at a previous annual meeting, and that printed copies of the suggested alteration or amendment shall have been circulated among the members, and that the members shall have been specifically advised that such alteration or amendment will be voted upon.

BY-LAWS

Article I.

SECTION 1.—These By-Laws shall merely interpret the Constitution and specifically apply its principles. They shall set forth no principles not included in the Constitution.

Article II.

SECTION 1.—All papers read before the Association shall become the property of the Association. Authors shall leave original copies of their manuscripts with the Editor or Reporter, at the time of presentation, for publication in the official journal.

SECTION 2.—When the number of papers makes it desirable, the Council may require authors to present their papers in abstract, and may set a time limit on discussions.

SECTION 3.—Members are urged to cooperate with all Scientific Committees of the Association.

SECTION 4.—Attendance at Annual Meetings and participation in the scientific programs shall be optional for all Honorary and Senior Members, but it shall be expected from all Active and Associate Members.

SECTION 5.—While the scientific session of the annual meeting is held primarily for the benefit of the members of the Association, it may be thrown open to nonmembers who are able to submit satisfactory credentials, who register in a specified manner, and who pay such registration fee as may be determined and published by the Council from year to year.

Article III.

SECTION 1.—Candidates for membership in this Association must be formally nominated and seconded, in an approved manner, by not less than three Active or Senior Members. Such nomination must have been in the hands of the Membership Committee for not less than four months, and the name of the candidate must have been distributed to the Association as a whole before final action may be taken on any new candidate for election to Active or Associate Membership. Provided the foregoing requirements have been met, and the candidates have been approved by the Membership Committee and by the Council, their names shall be presented to the Association at a regularly convened annual meeting for final action. A three-fourths vote of those present and voting shall be required to elect. Any candidate for membership

in this Association who has failed of election for two successive years shall automatically cease to be a candidate and may not be renominated until after a lapse of three years.

SECTION 2.—There shall be no numerical limit on Active Membership. The candidate, to be eligible, must be a citizen of the United States of America or of Canada, unless in unusual cases this citizenship requirement shall have been waived by the Council. The candidate shall have achieved distinction in the thoracic field, or shall have made a meritorious contribution to knowledge pertaining to thoracic disease or its surgical treatment. Under usual circumstances, Active Members shall be elected from the Associate Members who have satisfied all requirements for Active Membership. Under unusual circumstances, Active Membership may be awarded to new candidates of outstanding distinction.

SECTION 3.—There shall be no numerical limit on Associate Membership. The candidate, to be eligible, may be a citizen of any country. All candidates for Associate Membership shall be engaged actively in the practice of clinical medicine, or in teaching, or investigative work in the basic sciences underlying clinical practice, and all shall have demonstrated an active interest in thoracic disease. In the case of those who are engaged in the independent practice of thoracic surgery, it shall be required that they present evidence of sound basic training in general as well as thoracic surgery. Clinicians of outstanding ability, whose interest is limited to a small fraction of the entire thoracic field, may be elected to Associate Membership.

SECTION 4.—The number of Senior Members shall be unlimited. The candidate, to be eligible, must have been an Active Member of the Association for not less than ten years or, having been an active Member, have passed the age of sixty years, or become incapacitated by illness.

SECTION 5.—Honorary Membership shall be reserved for such distinguished persons as may be deemed worthy of this honor by the Council with concurrence of the Association.

SECTION 6.—The report of the Membership Committee shall be rendered at the annual Executive Session of the Association. Candidates shall be presented in groups in the following order: Candidates for Honorary Membership; retirement of Active Members to Senior Membership; candidates for Active Membership; candidates for Associate Membership.

SECTION 7.—The Council shall recommend that any Active or Associate member whose dues are in arrears for two years, or who has been absent, without sufficient excuse, from three consecutive annual meetings, shall have his membership terminated.

SECTION 8.—Notwithstanding Section 7, any member of the Association over 60 years of age is excused from the attendance requirement and upon his specific request may likewise be excused from the payment of dues.

ARTICLE IV.

SECTION 1.—The President of the Association shall perform all duties customarily pertaining to the office of President. He shall not only preside at all

meetings of the Association, but also at all meetings of the Council. The President shall be elected from the Active Members of the Association.

SECTION 2.—The Vice-President of the Association shall perform all duties customarily pertaining to the office of the Vice-President, not only as to the Association, but also as to the Council. The Vice-President shall be elected from the Active Members of the Association.

SECTION 3.—The Secretary of the Association shall perform all duties customarily pertaining to the office of Secretary. He shall serve not only as Secretary of the Association but also as Secretary of the Council. The Secretary shall be elected from the Active Members of the Association.

SECTION 4.—The Treasurer of the Association shall perform all duties pertaining to the office of Treasurer. He shall not only serve as Treasurer of the Association but shall also serve as custodian of the Endowment Fund. The Treasurer shall be elected from the Active Members of the Association.

SECTION 5.—The Editor of the Association shall be the Editor of the official Journal and shall, ex officio, be the Chairman of the Editorial Board. The Editor shall be elected from the Honorary, Active, or Senior Members of the Association.

SECTION 6.—The Councilors of the Association shall hold office as specified in the Constitution. They may be elected from either the Active Members or the Senior Members of the Association.

SECTION 7.—In the event of a vacancy occurring in the office of President, the Council shall advance the Vice-President to the Presidency and appoint a new Vice-President under the Provisions of Article IV, Section 3, of the Constitution.

ARTICLE V.

SECTION 1.—The Nominating Committee shall consist of three Active or Senior Members elected in accordance with the provisions of Article V, Section 1, of the Constitution. The Council shall instruct the Committee as to the vacancies which are to be filled by election.

SECTION 2.—The Membership Committee shall consist of seven Active or Senior Members appointed in accordance with the provisions of Article V, Section 2, of the Constitution. The Council may appoint not more than one of its own members to serve on this Committee. The duties of the Membership Committee are to investigate all candidates for membership in the Association and to report their findings as expeditiously as possible to the Council through the Secretary of the Association. This Committee is also charged with searching the literature of this and other countries to the end that proper candidates may be presented to the Association for consideration. Appointment to this Committee shall be for a period of one year, and not more than five of the members may be reappointed to succeed themselves. This Committee is also charged with maintaining a record of membership attendance and participation

in the scientific programs and reporting to the affected members and to the Council any deviations from the requirement of Article II, Section 4, of these By-Laws.

SECTION 3.—The Auditing Committee shall consist of three Active or Senior Members appointed in accordance with the provisions of Article V, Section 2, of the Constitution. None of these may be selected from the officers or councilors of the Association. Their duty shall be to audit the accounts of the Association at the end of each fiscal year and render their report to the Council of the Association through the Secretary. Appointments to this Committee shall be made for a one-year term. Not more than two members may be reappointed to succeed themselves.

SECTION 4.—The Program Committee shall consist of five members: The President of the Association, the Secretary of the Association, the Editor of the Association, and two members at large, one of whom shall be resident at or near the place of annual meeting. The duties of this Committee shall be to arrange, in conformity with instructions from the Council, the scientific program for the annual meeting.

SECTION 5.—The Necrology Committee shall consist of one or more Active or Senior Members, and shall be appointed in accordance with the provisions of Article V, Section 2, of the Constitution. Appointments to this Committee shall be for a one-year term of office. Any or all members of this Committee may be reappointed to succeed themselves. The Council may, if it so desires, appoint one of its own members to serve as Chairman of this Committee. The duties of the Necrology Committee shall be to prepare suitable resolutions and memorials upon the deaths of all members of the Association and to report such deaths at every annual meeting.

SECTION 6.—The Editorial Board shall be appointed by the Editor, subject only to the approval of the Council. The Editor shall be, ex officio, the chairman of this board and shall be privileged to appoint and indefinitely reappoint such members of the Association, regardless of class of membership, and such nonmembers of the Association as in his opinion may be best calculated to meet the editorial requirements of the Association.

SECTION 7.—When Scientific or Research Committees are authorized by the Association, the Council shall appoint the Chairmen of these Committees, with power to organize their committees in any way best calculated to accomplish the desired object, subject only to the approval of the Council. Financial aid rendered to such Committees shall not exceed such annual or special appropriations as may be specifically voted for such purposes by the Association as a whole.

ARTICLE VI.

SECTION 1.—Honorary Members of the Association are exempt from all initiation fees, dues, and assessments.

SECTION 2.—Annual dues for Active Members shall be $30.00.

SECTION 3.—Annual dues for Associate Members shall be $25.00.

SECTION 4.—Senior Members are exempt from dues.

SECTION 5.—Initiation fee for those elected directly to Active Membership shall be $15.00.

SECTION 6.—Initiation fee for those elected to Associate Membership shall be $10.00. If and when an Associate Member is elected to Active Membership, he shall pay an additional $5.00 initiation fee.

SECTION 7.—Income from the Endowment Fund shall be expended as the Council directs.

SECTION 8.—Associate and Active Members must subscribe to THE JOURNAL OF THORACIC SURGERY to retain their membership status.

SECTION 9.—Senior Members may retain their membership status without the payment of annual dues and subscription to THE JOURNAL OF THORACIC SURGERY is optional.

(Note.—Bills for membership dues and for subscriptions to THE JOURNAL OF THORACIC SURGERY *will be mailed to members by the Treasurer after the Annual Meeting.*)

ARTICLE VII.

SECTION 1.—When the Association convenes for its annual meeting, it shall immediately go into executive session, but the business at this session shall be limited to:

1. Election of Nominating Committee.
2. Appointment of necessary committees.
3. Miscellaneous business of an urgent nature.

SECTION 2.—The annual executive session of the Association shall be held at the opening of the afternoon session of the second day of the meeting. The order of business shall be:

1. Reading of the minutes of the preceding meetings of the Association and Council.
2. Report of the Treasurer for the last fiscal year.
3. Report of the Auditing Committee.
4. Report of the Treasurer for the current year to date.
5. Report of the Necrology Committee.
6. Report of the Program Committee.
7. Action on amendments to the Constitution and By-Laws.
8. Action on recommendations emanating from the Council.
9. Unfinished Business.
10. New Business.
11. Report of the Membership Committee.
12. Election of new members.
13. Report of the Nominating Committee.
14. Election of officers.

SECTION 3.—At the annual meeting of the Council the order of business shall be:

1. Reading of minutes.
2. Treasurer's report.

3. Appointment of committees.

4. Determination of instructions to be given to Auditing, Membership, Necrology, and other committees except Program Committee.

5. Determination of details in connection with the annual meeting.

6. Appointment and instructions to the Program Committee.

7. Unfinished Business.

8. New Business.

SECTION 4.—There shall be an annual meeting of the Council.

ARTICLE VIII.

SECTION 1.—These By-Laws shall in no wise be changed except by a two-thirds vote of the members present at the annual meeting or a properly convened meeting of the Association, and further provided that the proposed action or amendment shall have been moved and seconded by not less than three of the members in a properly convened annual or special meeting of the Association.

SECTION 2.—These By-Laws may be suspended in whole or in part for a period of not more than twelve hours by a unanimous vote of those present at any regularly convened meeting of the Association.